"*I would have everie man write what he knowes and no more.*"—MONTAIGNE

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EDITORIAL

THERE is reason to wonder whether any single specialty in medicine has undergone, during the past ten years, so rapid a development, in such a short time, as anaesthesia. This evolution is reflected in the interest of members of the specialty in their subject, even in its more academic aspects. Before the war, the section of anaesthetists of the Royal Society of Medicine used to meet in the smaller West Hall, and numbered 110. Since 1946 the section has occupied the Barnes Hall and now numbers 325.

In addition to the ever increasing numbers of Fellows and Members of the Association of Anaesthetists (307 Fellows, 730 Members in 1954), and the inception and healthy growth of the Faculty, the supreme academic authority in anaesthesia in this country, this interest is manifested by the very active societies which now exist in each region. We have from time to time published forthcoming programmes of some of these societies, and they are eloquent witness to their strength and enterprise. Unfortunately, it is rarely convenient or indeed possible for provincial anaesthetists either of consultant or more junior status to attend the meetings of the Royal Society of Medicine. Indeed, to come from the far North for such a meeting is often out of the question from the point of view of either finance or time. The local societies must therefore be the main Forum of opinion for the greater number of our anaesthetists. Furthermore these local societies encourage trainees to report on their experiences and thereby assist in the development of clinical observation and research. The *British Journal of Anaesthesia* is anxious to give every encouragement possible to such activities. A new step was the publication in our last issue of the proceedings of a meeting of the Society of Anaesthetists of the South-Western Region, and in this issue we are giving a similar report, including a discussion, of the Liverpool Society of Anaesthetists. This latter is the oldest provincial Society of Anaesthetists, being founded in 1931. It has on its strength 75 members and meets monthly during the season which exists from October to May. Next year it is to celebrate the 25th anniversary of its foundation.

We would be very pleased to receive similar reports of the meetings of other societies. Very excellent papers are read at these meetings and not infrequently are followed by interesting and stimulating discussions. It seems a great shame that these should not be reported and made available to as wide a public as possible.

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CARBON DIOXIDE CONCENTRATIONS IN THE BOYLE'S TYPE ANAESTHETIC MACHINE*

BY

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THE elimination of carbon dioxide and the concomitant regulation of the pH of the blood is a respiratory function no less important than the uptake of oxygen. The elimination of carbon dioxide from anaesthetic atmospheres and the concentrations of this gas normally reached in such atmospheres are problems which are engaging a considerable amount of attention at the present time.

Schwartz, Alexander and Adriani (1953) have studied this problem for varying gas flows in the American type of anaesthetic machine. We were unable to trace any work of a similar nature using the standard British Boyle's pattern anaesthetic machine, nor could the manufacturers of our machines supply us with experimental data. Molyneux and Pask (1951) have estimated the volume of backflow of expired gases to the reservoir bag in a semi-closed circuit under certain theoretically deduced conditions, and we have therefore investigated the actual rise in carbon dioxide concentrations in the same type of semi-closed circuit.

Ringrose, Rowling and Harbord (1950) have developed a carbon dioxide indicator designed to make quantitative measurements of this gas in anaesthetic atmospheres. Extensive trials with this R.R.H. Indicator failed to give us reproducible analyses, and we were unable to demonstrate any correlation between the values determined by this instrument and the values determined for the same gas samples when they were analyzed in the Haldane apparatus. Since no calibration curve could be constructed, use of the R.R.H. Indicator had to be abandoned.

EXPERIMENT

Using a standard Boyle's type anaesthetic machine, oxygen was delivered to conscious adult male volunteers at a flow rate of 8 l./minute *via* a corrugated tube of capacity ± 650 ml and a Connell type mask fitting accurately to the subject's face (fig. 1). The reservoir bag of the circuit had a capacity of half a gallon (2,273 ml). The same machine was used in each experiment and gas sampling points were provided proximally at a point just distal to the reservoir bag (A) and distally at the mask itself (B). These sampling points are indicated in figure 1.

The subjects were allowed to breathe from this apparatus for several minutes. During the sixth minute of breathing, two 10-ml gas samples were drawn from each of the two sampling points. Sampl-

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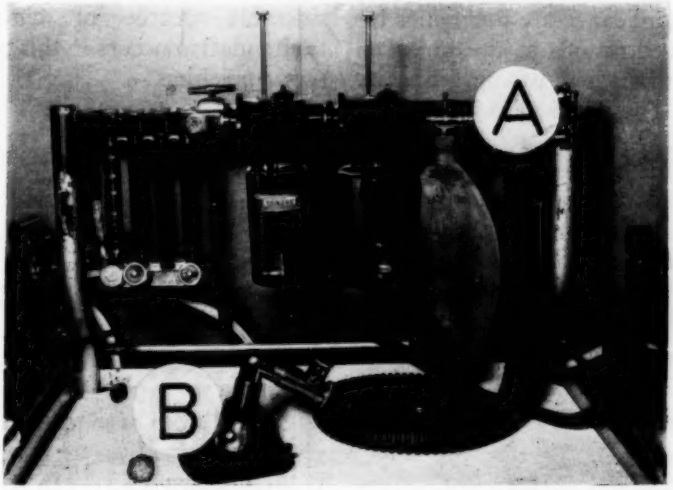

FIG. 1
The type of anaesthetic machine which was used in this investigation. The two sampling points are indicated by the letters.

ing was achieved during the expiratory phase of several breaths, the samples being drawn into all-glass syringes lubricated with glycerine. The syringes were then fitted with gas-tight caps until their contents could be analyzed in duplicate on a standard Haldane apparatus by the usual technique (Lamb, 1930). The values given in table I represent the arithmetical means of duplicate analyses.

TABLE I

Experiment number	Volumes per cent carbon dioxide	
	At mask	At reservoir bag
1	5.12	1.78
2	4.63	0.11
3	4.70	0.33
4	5.11	0.22
5	5.21	0.22
6	5.07	0.17
7	5.39	1.21
8	4.78	0.49
9	5.27	0.38
10	5.00	0.16
11	5.16	0.74
Mean	5.04	0.53
S.E. of Mean	±0.05	±0.02
P	<0.01	<0.01

RESULTS

The concentration of carbon dioxide in atmospheric air is of the order of 0.03 vol. per cent (NTP) ($pCO_2 = 0.23$ mm Hg) while the concentrations in alveolar air are accepted as being of the order of ± 6.0 vol. per cent ($pCO_2 = 45.6$ mm Hg) under normal conditions. We performed 13 experiments on 10 subjects but in 2 experiments the subjects began hyperventilating. This hyperventilation, which may of itself be significant, produced values, at the reservoir bag, which were 8 times greater than the mean values for the CO_2 concentration for the whole series. The results of these two experiments have been discarded and the results of the remaining 11 are set out in table I.

In view of the increased dead-space produced by the mask, the values for the CO_2 concentrations at the mask (mean: 5.04 vol. per cent = 38.3 mm Hg pCO_2) are to be expected.

Of more significance are the values for the CO_2 concentrations at the proximal end of the corrugated tubing (capacity ± 650 ml). The mean of 0.53 vol. per cent CO_2 (pCO_2 = 4.03 mm Hg) is 17 times the value for room air. At this sampling point the value depends not only on the gas flow rate from the machine but also on the respiratory minute volume. It is apparent that under the conditions of these experiments a considerable degree of carbon dioxide accumulation occurs in this type of anaesthetic machine.

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ACQUIRED TOLERANCE TO INTRAVENOUS THIOBARBITURATES IN ANIMALS

BY

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SEEVERS (1954) has defined tolerance to narcotics as "the partial or complete immunity to quantities of these substances which would otherwise diminish or completely abolish the functional activity of certain neurophysiological and other mechanisms." The repeated administration of medium-acting barbiturates leads to the development of tolerance to their narcotic effects. This applies to amylobarbitone and isopropyl-β-bromallyl-barbiturate in rabbits (Fitch, 1930) and delvinal and pentobarbitone in guineapigs and rats (Carmichael, 1941, 1948). The above workers and Gruber and Keyser (1946) have convincingly demonstrated that tolerance to one barbiturate produces a cross tolerance to other drugs of the same series.

Mice develop a tolerance to the daily administration of thiopentone. This effect is maximal in five to six days and amounts to a decrease in sleeping time by about fifty per cent (Hubbard and Goldbaum, 1949). Thrice weekly administrations of thiopentone to the dog lead to a forty per cent decrease in the duration of sleep by the end of the third week (Dundee, 1953). Green and Koppanyi (1944) found that dogs rendered tolerant to thiopentone showed cross tolerance to hexobarbitone and *vice versa*. The author has verified this with thiopentone, thialbarbitone, thiamylal and hexobarbitone.

In the course of clinical work the author has encountered several cases of tolerance to thiobarbiturates, in which the patients had been receiving large doses of sedative or analgesic drugs. In none of these patients could the resistance be attributed to acquired tolerance to medium-acting barbiturates. Similarly the drugs being administered to each of the resistant patients varied daily, but in each case they were limited to one type of drug such as D.D.A. analgesics or aspirin-like analgesics.

Experiments were carried out in dogs and rats to see if the daily administration of one type of sedative or analgesic did, in fact, increase the tolerance to thiobarbiturates. All thiobarbiturates were given by the intraperitoneal route, and during the experiment the interval between these injections was such as to avoid tolerance from the drugs themselves. Several control readings were done before the daily administration of sedatives or analgesics was started. All medication was stopped for 24 hours before injection of the thiobarbiturates, but drugs were frequently given when the animals wakened. At the

TABLE I

Effect of increasing doses of analgesics on the duration of narcosis with intraperitoneal injection of 30 mg/kg thiopentone and thiamylal in the dog. Each reading is the average of 4 observations. Unless stated above all analgesics were administered per os.

Days	Drugs administered	Narcosis			
		Thiopentone		Thiamylal	
		Onset (min)	Duration (min)	Onset (min)	Duration (min)
	Thiobarbiturate control (average of 4)	7.0	42	5.2	56
1	Morphia 15 mg (I.M.) Amidone 20 mg				
2	Pethidine 100 mg : Phenadoxone 60 mg				
3	Levorphan 12 mg				
4	Amidone 20 mg : Levorphan 4 mg (I.M.) Dilaudid 2 mg (I.M.) : Hyperduric morphia 32 mg (I.M.)				
5	Hyperduric morphia 65 mg (I.M.)				
6	Morphia 15 mg (I.M.) : Phenadoxone 60 mg				
7	Pethidine 600 mg + 100 mg (I.M.)				
8	*Thiobarbiturates* ... ... Levorphan 3 mg	10.2	14.0	6.0	52.1
9	Amidone 20 mg : Morphia 32 mg (I.M.) Pethidine 100 mg (I.M.) : Amidone 10 mg (I.M.)				
10	Hyperduric morphia 100 mg (I.M.)				
11	Amidone 15 mg : Dilaudid 4 mg (I.M.) Pethidine 100 mg (I.M.)				
12	*Thiobarbiturates* ... ... Phenadoxone 30 mg	7.0	21.2	8.0	29.0
13	Levorphan 9 mg				
14	Amidone 50 mg				
15	Levorphan 9 mg : Morphia 16 mg (I.M.) Dilaudid 2 mg (I.M.)				
16	Amidone 60 mg : Levorphan 2 mg (I.M.)				
17	*Thiobarbiturates* ... ... Levorphan 3 mg	10.2	5.0	12.0	24.5
18	Levorphan 15 mg : Pethidine 100 mg Pethidine 100 mg (I.M.)				
19	Hyperduric morphia 100 mg (I.M.) Pethidine 100 mg (I.M.)				
20	Amidone 70 mg				
21	Pethidine 600 mg : Amidone 30 mg ...				
22	*Thiobarbiturates* ... ... Levorphan 6 mg	11.0	16.0	8.2	23.2
23	Amidone 80 mg				
24	Hyperduric morphia 128 mg (I.M.)				
25	Levorphan 15 mg + 4 mg (I.M.)				
26	Pethidine 600 mg : Phenadoxone 60 mg				
27	Amidone 100 mg				
28	*Thiobarbiturates* ... ... Levorphan 3 mg	—	0	9.0	12.5
29	Morphia 64 mg (I.M.) : Pethidine 100 mg				
30	Amidone 40 mg				
31	Amidone 20 mg : Levorphan 3 mg				
32	Amidone 10 mg : Levorphan 1.5 mg				
33	Morphia 16 mg (I.M.)				
35	*Thiobarbiturates*	7.0	51	6.2	75
40	*Thiobarbiturates*			6.0	66
45	*Thiobarbiturates*	7.0	60	4.0	56

TABLE II

Effect of increasing doses of analgesics on the duration of narcosis with 60 mg/kg thialbarbitone in 32 rats. All analgesics were given by I.M. injection

Days	Drugs administered to each animal	Narcosis	
		Per cent asleep	Average duration (min)
0	Control (average of 3 readings)	81	18.4
1	Pethidine 5 mg/kg		
2	Levorphan 0.1 mg/kg		
4	Levorphan 0.2 mg/kg		
7	Levorphan 0.2 mg/kg		
8	Levorphan 0.2 mg/kg : Amidone 1 mg/kg		
9	Morphia 1.5 mg/kg : Amidone 1 mg/kg		
10	*Thialbarbitone*	75	6.2
11	Morphia 2.2 mg/kg : Pethidine 15 mg/kg		
12	Morphia 4.4 mg/kg		
14	Morphia 2.2 mg/kg : Pethidine 15 mg/kg		
15	—		
16	Levorphan 0.3 mg/kg : Pethidine 15 mg/kg		
17	*Thialbarbitone*	67	1.9
18	Morphia 2.2 mg/kg : Pethidine 15 mg/kg		
19	Pethidine 30 mg/kg		
21	Levorphan 0.45 mg/kg : Morphia 2.2 mg/kg		
22	Levorphan 0.67 mg/kg		
23	Levorphan 0.45 mg/kg : Morphia 3.3 mg/kg		
24	*Thialbarbitone*	0	—
25	Levorphan 0.45 mg/kg : Pethidine 30 mg/kg		
26	Levorphan 0.9 mg/kg		
28	Pethidine 20 mg/kg : Levorphan 0.45 mg/kg		
29	Pethidine 40 mg/kg : Levorphan 0.45 mg/kg		
30	Pethidine 40 mg/kg : Morphia 4.4 mg/kg		
31	*Thialbarbitone*	0	—
37	*Thialbarbitone*	73	13.1

end of the procedure inert placebo tablets were given and the anaesthetic repeated several times at intervals of five to six days.

RESULTS

The average effect of the daily administration of increasing doses of potent analgesics on the duration of action of three thiobarbiturates is shown in tables I and II. In each of these series there was a gradual reduction in the duration of narcosis with the thiobarbiturates, but within five days of stopping the analgesics the duration of action of the narcotic returned to normal. Table III shows that the less potent analgesics of the aspirin type produced a similar although less marked effect.

In table IV is shown the average effect of the daily administration of non-barbiturate sedatives on the duration of action of thiopentone in the dog. These drugs likewise induce tolerance to thiobarbiturates which is as marked as that from the D.D.A. analgesics. Anti-histamines of varying types were given to the animals of table V, and again there was a gradual shortening of the action of the thiobarbiturate, although not as marked as with the other drugs studied. Chlorpromazine similarly induces a mild degree of tolerance to thiobarbiturates.

TABLE III

The effect of repeated oral administration of mild analgesics on the duration of narcosis following intraperitoneal injection of 30 mg/kg thiopentone and thiamylal in the dog. Each reading is the average of 4 administrations.

Day	Drugs administered to each animal	Narcosis			
		Thiopentone		Thiamylal	
		Onset (min)	Duration (min)	Onset (min)	Duration (min)
	Thiobarbiturates control (average of 5)	8.0	46.2	4.0	86.2
1	Aspirin 325 mg : Phenacetin 400 mg				
2	Aspirin 650 mg : Aspirin & Dovers 2 tabs.				
3	Aspirin 650 mg : Aspirin & Dovers 2 tabs.				
4	Aspirin 1950 mg : Aspirin & Dovers 6 tabs.				
5	Tabs. Codeine Co. 12 tabs.				
6	Saridone 4 : Mephosol 3 tabs.				
7	Aspirin & Dovers 4 : Mephosol 2 tabs.				
8	Cefonin 6 tabs.				
9	Mephosol 3 tabs. : Aspirin 1950 mg				
10	*Thiobarbiturates* ... ... Tabs. Codeine Co. 3 tabs.	11.0	11.0	6.0	60
11	Neurodyne 4 tabs. : Aspirin 925 mg				
12	Neurodyne 4 tabs. : Tabs. Codeine Co. 4 tabs.				
13	Nephosol 6 tabs.				
14	Aspirin & Dovers 3 tabs. : Tabs. Codeine Co. 4 tabs.				
15	*Thiobarbiturates*	9.2	25.0	7.8	56
16	Nephosol 3 tabs.				
16	Arthipax 7 tabs.				
17	Aspirin 2600 mg				
18	Mephosol 8 tabs.				
19	Aspirin 2600 mg.				
20	*Thiobarbiturates* ... ... Mephosol 2 tabs.	12.0	10.3	10.0	45.5
21	Mephosol 6 tabs. : Aspirin & Dovers 2 tabs.				
22	Aspirin 1040 mg : Tabs. Codeine Co. 4 tabs.				
23	Mephosol 8 tabs.				
24	Aspirin 3300 mg				
25	*Thiobarbiturates* ... ... Mephosol 6 tabs.	10.0	4.5	9.0	33.5
26	Aspirin 3900 mg				
27	Mephosol 10 tabs.				
28	Neurodyne 8 tabs.				
29	Cefonin 4 tabs. : Saridone 6 tabs.				
30	*Thiobarbiturates*	—	0	9.0	38
31	Aspirin & Dovers 4 tabs.				
32	Tabs. Codeine Co. 2 tabs.				
33	Mephosol 3 tabs.				
34	Aspirin & Dovers 2 tabs.				
35	Mephosol 2 tabs.				
36	Mephosol 2 tabs.				
40	*Thiobarbiturates*	6.0	60.0	5.5	95.0
45	*Thiobarbiturates*	5.5	58.5	4.0	77

The analgesics referred to above contain the following per tablet:

Saridone (Roche)
- phenyl-dimethyl-isopropyl-pyarazolon 150 mg
- acetiphenetidin 250 mg
- caffein 50 mg

Aspirin & Dovers (Tabs. Acetylsalicyli c Ipicac et Opio. N.F.)
- Acetylsalicylic acid 162 mg
- Pulv Ipecac et Opio 162 mg

Neurodyne (Wade)
- Acetylsalicylic acid 260 mg
- Calcium Carbonate 65 mg
- Magnesium Carbonate 25 mg
- Phenacetin 195 mg
- Codeine phosphate (B.P.) 6.5 mg

Tabs. Codeine Co. (N.F.)
- Codeine phosphate 8 mg
- Acetylsalicylic acid 260 mg
- Phenacetin 260 mg

Cefonin (Honmel)
- Phenylsemicarbazide 260 mg
- Caffein 42 mg
- Lactose 325 mg

Arthipax (Clinical Products Ltd.)
- Mephenesin 65 mg
- Salicylamide 325 mg
- Dihydroxyaluminium glycinate 100 mg

Mephosol (Crooks)
- Mephenesin 125 mg
- Salicylamide 250 mg
- Homatropine methyl bromide 0.4 mg

TABLE IV

Average effect of daily oral doses of non-barbiturate sedatives on the duration of action of thiopentone (30 mg/kg) in the dog. Each reading is the average of 3 observations.

Days	Drugs administered	Narcosis	
		Onset	Duration
0	Thiopentone control (average of 3)	6.3	53
1–10	Sedatives		
11	Thiopentone	6.0	35
12–16	Sedatives		
17	Thiopentone	7.0	12
18–25	Sedatives		
26	Thiopentone	—	0
27–30	Sedatives		
31	Thiopentone	10.0	40
40	Thiopentone	7.0	51

Sedatives included:
K Br, Chloral hydrate, Rhysoval, Bromisovalerylurea, Carbromalum, Chloretone, Methylpentynol, Persodon

TABLE V

Effect of daily oral administration of increasing doses of various anti-histamines or thiamylal narcosis (30 mg/kg) in the dog. Each reading is the average of 4 observations.

Days	Drugs administered	Narcosis	
		Onset	Duration
0	Thiamylal control (average of 3)	4.7	74.2
1–8	Anti-histamines		
9	Thiamylal	5.5	55.0
10–15	Anti-histamines		
16	Thiamylal	6.0	48.0
17–23	Anti-histamines		
24	Thiamylal	6.2	45.0
25–30	Inert placebos		
31	Thiamylal	5.0	66.0
36	Thiamylal	4.0	67.0

Anti-histamines administered include:
Phenergan, Dibistin, Ancoran, Synopen, Antistin, Dramamine, Theophrin and Benadryl.

DISCUSSION

Many suggestions have been offered to explain acquired tolerance to narcotics. These include increase in rate of detoxication of the drugs, interference with chemical mediators or hormones by repeated doses of the drugs and enzyme adaptation. None of the aforementioned hypotheses has been confirmed. Hubbard and Goldbaum (1949) found that mice rendered tolerant to the daily administration of thiopentone awaken at higher tissue levels than controls. The above results show that the onset of anaesthesia was delayed after intraperitoneal injection of thiopentone and thiamylal in dogs who had received large doses of analgesics or narcotics. These findings suggest that tolerance is an adaptation of the nervous system to higher thiobarbiturate levels.

The narcotic effects of morphine (and similar drugs) have been explained by a combination of the drug with receptors near, or on the surface of the neurone. Tolerance consists of a maximal, but never complete saturation of these receptors comparable to that postulated for tachyphylaxis with other agents. Cross tolerance to drugs with a similar chemical

TABLE VI

Effect of daily administration of chlorpromazine on narcosis induced by intraperitoneal injection of 30 mg/kg thiamylal in the dog. Daily doses of chlorpromazine started at 4–5 mg/kg by oral administration and gradually increased to 12–15 mg/kg.

Days on chlorpromazine	No. of observations	Average total dose of chlorpromazine given		Average narcosis in minutes	
		mg/kg	mg	Onset	Duration
0	12	—	—	4.5	54 ± 7
17	4	120	1500	6.0	45 ± 9.5
24	4	190	2180	7.0	29 ± 0
29	4	240	2880	7.0	38 ± 7.0
39	4	290	3480	6.5	43 ± 12.5
52	4	445	5340	7.0	36 ± 4.7
4 days after stopping chlorpromazine	4	—	—	5.0	60

structure can be explained by similarity of the "anchoring groups". This does not explain cross tolerance between compounds of entirely dissimilar chemical characteristics such as thiobarbiturates and opiates. Seevers (1954) has suggested that this is related to the increase in latent hyperexcitability of the neurone rather than to any specific competitive effect.

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ANAESTHESIA FOR CHILDREN, WITH SPECIAL REFERENCE TO TONSILLECTOMY BY DISSECTION

A Report on 295 Cases

BY

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WHEN one reviews the literature on the subject of anaesthesia for children, the themes of safety for the child and simplicity of technique become almost inseparable.

The advent of the thiopentone-gallamine-nitrous oxide-oxygen sequence, followed by intermittent pethidine, for almost any operation in adults, has shown us that the human subject can be anaesthetized and controlled in a very light plane of anaesthesia with great ease and simplicity, and with remarkably few post-operative complications. It seemed reasonable, therefore, to reconsider the anaesthetic techniques for operations on children to which anaesthetists have been clinging because in the main they were considered to be either safe or simple, or both.

At operation for dissection of tonsils and adenoids Hawksley (1945) prefers open ethyl chloride and ether taken to the third plane of stage three of anaesthesia, using a premedication of rectal paraldehyde 1 drachm per stone (4 ml/6.4 kg) in 10 per cent solution. Slater and Stephen (1951) prefer a minute dose of premedication and declare there is no excuse for using such a dangerous drug as ethyl chloride when vinyl ether (Vinesthene) is available. Maintenance should be with nitrous oxide-oxygen-ether after endotracheal intubation in every case regardless of the age and size of the patient. These workers are supported by Eather (1952) who believes that endotracheal anaesthesia is essential for safety and success. Jarvis (1953) uses small doses of pentobarbital (Nembutal) and hyoscine as premedication; induction is with vinyl ether and open ether over an average period of ten minutes to allow for difficulties with swallowing and breath holding.

Certain desiderata in anaesthesia for children such as simplicity of equipment, a readily controllable dosage, and a technique which could be employed by an anaesthetist without the help of an assistant, have hitherto been regarded as fundamental. These have been deliberately laid aside by the author with the idea of clearing the field for a trial of a method which would bring the anaesthetic as near the adult standard of pleasantness and safety as possible.

The "Open" or "Rag and Bottle" method requires a very simple apparatus, but it delivers a variable dosage of highly toxic vapours such as ethyl chloride, ether, or chloroform, and results in

unpleasant and nauseous complications. There is a not infrequent incidence of stormy inductions with screaming, fighting children and periods of breath holding, spasm, and resultant cyanosis. Such an induction may sometimes be remembered by the little patient afterwards. The stimulus to memory, however, may only occur when the patient is prepared in a similar manner for some subsequent operation.

The question of management by a single-handed anaesthetist is of importance, but it must now be very rare to find circumstances which compel an anaesthetist to anaesthetize a child absolutely without assistance. Taking for granted that there would be someone to hand, a different technique was tried out very tentatively with the staff fully realizing that there might be difficulties which had not been foreseen, and which might need prompt and intelligent action.

PREMEDICATION

In order to keep in line with the technique for adults, the children were premedicated with papaveratum (Omnopon) and scopolamine, given one and a half hours pre-operatively, in doses (table I) which conform with the scale described by Anderson (1951).

Such premedication has been criticized in that the children are often not asleep at induction. Experience has shown that although not asleep the children are quiet and co-operative. At East Grinstead the combination of papaveratum and scopolamine has not been found as satisfactory a premedication as that of quinalbarbitone (Seconal) 0.6 gr per st (6 mg/kg) of body weight to a maximum of 3 gr (192 mg) and atropine 1/100 gr (0.65 mg). The latter drugs almost invariably provide a sleeping child at induction. It is conceded that, with any regime, bad timing and inaccurate dosage of premedicant drugs will tax the ingenuity and patience of the most skilful anaesthetist.

TABLE I
Dosage of premedicant drugs

lb	kg	Papaveratum gr	Papaveratum mg	Scopolamine gr	Scopolamine mg
28	12.6	1/9	7.0	1/450	0.14
32	14.5				
36	16.3				
40	18.1				
42	19.0	1/6	11.0	1/300	0.22
46	20.9				
50	22.7				
54	24.5				
56	25.4	1/4	16.0	1/200	0.32
60	27.2				
64	29.0				
68	30.9				
70	31.6	1/3	21.0	1/150	0.43
74	33.6				
78	35.4				
82	37.2				
84	38.1				

INDUCTION OF ANAESTHESIA

In most cases under the age of about seven years the induction of anaesthesia was started with a game, for the patient was invited to blow up a balloon—the reservoir bag. During this "game" nitrous oxide flowed at 10 l. per minute into the reservoir bag and the child blew back into the bag through the face-piece and the corrugated rubber hose. After two or three breaths, cyclopropane was added at 700 ml per minute, and oxygen at 2 l. per minute. The nitrous oxide flow was reduced to 4 l. per minute and an assistant then took over the face-piece. The cyclopropane was turned off and thiopentone and gallamine administered together intravenously. Cyclopropane was

employed in order to avoid cyanosis and provide good conditions for the injection.

The dosage of thiopentone presented a problem, but was estimated according to weight. Anderson (1951) has pointed out the marked variation in weight for age in young patients. Bullough (1952) appears satisfied with a less scientific estimation using age alone, which he states is satisfactory in his hands. Trials were carried out in order to assess the dosage on a weight and age basis. It was found that a satisfactory dose could be computed if the weight alone was used as the standard, and 12 mg of thiopentone given for each kg of body weight. From 10 to 15 mg per kg could be used, according to the variation in weight for age, but no fixed rule was possible for this calculation. Anderson (1951) gave the following weight for age variations.

No. of cases	Age	Weight (lb)
58	4	28–50 lb (12.6–22.7 kg)
55	8	44–92 lb (20.0–42.5 kg)
33	12	66–112 lb (30.0–50.8 kg)

At the beginning of the series approximately 2 mg/kg gallamine triethiodide were used; so that for a child of six years, weighing 25 kg (about 4 st), 300 mg (6.0 ml of 5 per cent solution) of thiopentone with 50 mg of gallamine would be given. Such a dosage almost invariably produced apnoea for a short period. As soon as spontaneous respiration was re-established the surgeon was allowed to put on the towels and begin the operation. The complete relaxation provided for the insertion and opening of the Boyle-Davis gag was a great advantage. A slight pause was usually made just after opening the gag, in order to be quite certain that the patient's tidal volume was really adequate before surgery was begun. Should the timing of the return of an adequate tidal volume have been misjudged it was found quite simple to ventilate the child by gentle rhythmical compression of the chest. For this the anaesthetist's hand was placed underneath the sterile towels, and it was not found necessary to disturb the surgical preparations for operation. Employing thiopentone and a relaxant the airway was not a problem, but the adequacy of the respiration had to be watched most carefully. In the one case described in which there was a persistent apnoea, the child was over eight years of age and was therefore intubated on induction. After this induction a mixture of nitrous oxide (4 l.) and oxygen (2 l.) was supplied from the anaesthetic machine through the tube on the Boyle-Davis gag.

MAINTENANCE OF ANAESTHESIA

A Gordh needle was inserted into the vein in the back of the hand or foot, and pethidine administered as necessary. This also provided a ready route for the injection of resuscitative drugs if required.

It was soon found in the earlier cases in the series that rather smaller doses of thiopentone, and slightly smaller doses of gallamine would suffice, provided the premedication had been well timed and the patient correctly assessed. The doses suggested above were drawn up into the syringe ready and, usually at the time of making the injection, the correct proportion of this amount to be used was estimated. This gave a fairly flexible range which soon became easy to handle. In the earlier cases the larger dose of drugs made it possible to maintain the

patient for the remainder of the operation on nitrous oxide and oxygen (2 l. : 1 l.). When, however, the dosage of thiopentone and gallamine was reduced it became evident that the dilution of the nitrous oxide and oxygen mixture by air in the nasopharynx resulted in too light an anaesthetic.

Some adjuvant was clearly needed in order to maintain satisfactory operating conditions. With the object of this investigation in mind, the obvious adjuvant to use was pethidine administered through the Gordh needle. The assessment of the dosage of pethidine to be given was influenced by the possibility of respiratory depression. Though children seem to metabolize drugs rapidly, it was appreciated that powerful depressants had been used both in premedication and induction. In 1951, no experience had been gained of such antidotes as nalorphine. Pethidine was, therefore, given in small doses of 5–10 mg which could readily be repeated if required. A dilute solution of pethidine of 10 mg/ml was used in order to facilitate the handling of these small doses.

Some difficulty was experienced with the need for supporting the Boyle-Davis gag. It appeared after trials of different methods that the most satisfactory, from the point of view of the surgeon, was for the anaesthetist personally to hold this gag. In order to be free to make the various injections it was obviously necessary that the assistant nurse should be shown how to support the gag while the anaesthetist administered the drugs. She could not be employed in this way for prolonged periods owing to her other duties in the theatre. It was astonishing how often instruments would fall on the floor just as the change-over had been negotiated satisfactorily.

It appeared, therefore, that pethidine as an intravenous adjuvant to gas-oxygen in these circumstances had both advantages and disadvantages. Its analgesic properties made the running of the case smooth and simple at very light planes of anaesthesia, but the difficulties which have been described were encountered and in the earlier cases a few patients suffered respiratory depression which gave rise to some anxiety. Furthermore it was considered that suppression of the laryngotracheal reflex might be a danger with an operation in the mouth.

To overcome the difficulties described in the use of pethidine it seemed reasonable to employ a non-irritant vapour such as trichlorethylene, to supplement the narcotic action of nitrous oxide.

ENDOTRACHEAL INTUBATION

In young children undergoing tonsillectomy, endotracheal intubation was considered unnecessary from the anaesthetic point of view, and indeed it might be potentially dangerous, as the nasal tube inevitably carries organisms from the infected adenoids down into the chest. The surgical opinion, however, was that such infection was autogenous and would have little or no effect on the patient. Certainly there have been only three cases with postoperative chest complications which have needed chemotherapy. All the patients between the age of eight and fourteen were intubated at the request of the surgeon.

Intubation was performed in apnoea, following the initial dose of thiopentone

and gallamine. At this hospital each operation took between twenty and thirty-five minutes from the start of induction to the return to bed. In the earlier cases, with the larger doses of relaxant, it was considered wiser to use atropine and neostigmine at the end of the procedure just prior to the removal of the adenoids; in those cases that were intubated the tube was withdrawn at this stage. Of the 348 cases reviewed, 53 were for removal of adenoids only and are excluded from these figures. Of 295 cases, 107 children were between the ages of eight and fourteen years, and all were intubated; 188 children were under the age of eight, and were not intubated unless their size and body weight made the surgical management easier and safer if a tube were passed. Of these latter, 23 were intubated.

No. of cases	Intubated	Not intubated
295	130	165

DISCUSSION OF TECHNIQUE

When investigating this technique it was appreciated that in some centres the operating time of four to six minutes for each case would make such a method difficult to handle, and render the use of relaxants dangerous. In these centres both the anaesthetic and the operation are conducted as very minor procedures. Only sleep-doses of anaesthetics are used, with little or no tissue saturation. The technique described here was not intended for such centres, but rather is it an effort to avoid prolonged or deep ether anaesthesia in young children undergoing minor operations, where the operating time is considerably longer.

It is also appreciated that such a technique would not prove suitable for undergraduate students in a teaching hospital, and indeed should only be handled by those who employ relaxants regularly.

Ether convulsions are now rarely encountered, but the administration of thiopentone with a relaxant obviates both the risk of ether convulsions and also the chance of thiopentone spasm. The technique has now been tried on children for a wide field of surgery other than tonsillectomy and has proved remarkably easy and successful. The limiting factor would seem to be the availability of a reasonable vein through which to administer the drugs. Once anaesthesia is induced it has been found that adequate veins can more readily be located. To this end the use of a simple tourniquet consisting of a strip of motor tyre inner tubing, eighteen inches long and one and a half inches wide, to which may be attached a Spencer Wells, has proved invaluable. It provides an even pressure round the circumference of the limb, and makes the demonstration of superficial veins considerably easier than with manual compression.

No increase of bleeding or difficulty from bleeding was noticed in this series of cases but elsewhere this technique has been blamed by surgeons for excessive bleeding, although the number of swabs required in such cases was not unusually large and did not provide objective evidence of this difficulty. The ward staffs were certainly in favour of the peaceful recovery of the children dealt with by this technique. It is difficult to prove that any particular method or drug increases bleeding. Clearly any technique might be falsely blamed if, during the management

of a case, the respiration was depressed sufficiently to allow a build-up of carbon-dioxide or hypoxia with resultant cyanosis and peripheral congestion.

COMPLICATIONS AND DISADVANTAGES

In this series of 295 patients there have been 29 cases in which trouble of some kind has been experienced. Of these 29 cases, 11 had prolonged apnoea immediately after the injection of thiopentone and gallamine. In one of these 11 the apnoea persisted for nearly forty minutes after the tonsils were removed. It was not easy to assess whether this was, in fact, an apnoea solely due to a drug, or due to the mismanagement of the anaesthetist. The child was probably over-ventilated with resultant acapnia, and the carbon dioxide never allowed to achieve a concentration which would stimulate the respiratory centre. Of the 18 remaining cases, 8 showed some degree of respiratory depression at the end of operation, and were therefore given atropine and neostigmine. They gave no further trouble. Finally, 10 cases gave concern back in the ward, and when seen by the anaesthetist correction of the airway proved all that was necessary without recourse to resuscitative drugs. Similar trouble could have been experienced with other forms of anaesthesia.

Most of the troubles occurred in the earlier part of the series, for experience has led to a better assessment of dosage. The children have given very little trouble at all from the postoperative nursing point of view and all have been satisfactory clinically.

Postoperative vomiting is always a difficult factor to assess as the anaesthetist depends almost entirely on nursing reports. Such reports tend to be inaccurate with the ever changing duty hours. In this series of 295 cases it is reported that 39 patients (13.2 per cent) vomited at least once. In another series of 147 similar cases anaesthetized with "open" ethyl chloride and ether, 63 patients (42.8 per cent) vomited.

The patients who have been questioned after the present series seem to remember very little except waking to find they had sore throats for a day or two, and have no fears about visiting the hospital again.

CONCLUSIONS

It would seem that this technique is reasonably safe, convenient for the surgeon and pleasant for the patient, although difficult to manage and complicated to handle. The nursing staff prefer it as the children do well, are rarely restless, and postoperative vomiting is minimal.

The method may be greatly simplified in cases for tonsillectomy if the Gordh needle can be avoided by slightly reducing the initial dosage of gallamine and thiopentone, and substituting very small quantities of trichlorethylene for the pethidine as an adjuvant to nitrous oxide and oxygen. Under these conditions no change of the individual holding the gag is necessary as no subsequent injections have to be made. Other operations on children, during which the hands of the anaesthetist are free, are very easily handled with the thiopentone-relaxant-nitrous oxide-oxygen-pethidine sequence. It is considered that such a technique is well worth any extra trouble it may be to the anaesthetist.

SUMMARY

An adult type of technique for anaesthesia for children has been described with special reference to tonsillectomy.

Difficulties and dangers have been pointed out, and the time factor has been stressed.

The technique is not advised for those learning anaesthesia.

Even in prolonged cases the children need not be subjected to deep anaesthesia with its very undesirable consequences.

The subject is controversial, and has been re-opened in the hope that it may stimulate thought and discussion.

ACKNOWLEDGMENTS

I should like to thank Mr. R. Glennie, who operated on all these cases, for his patience and extremely helpful co-operation; and the sisters and nursing staff of the Homeopathic Hospital, Tunbridge Wells, who helped to make such a study possible.

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GENERAL ANAESTHESIA FOR A MORPHINE ADDICT

BY

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THE established drug addict presents the anaesthetist with an interesting problem when a major operation is proposed. As few references to this are made, either in the literature or in the text-books, it is thought that the following account of the management of such a case will be of interest.

CASE HISTORY

The patient, an unmarried woman, aged 54 years, was admitted to hospital on 11.4.54. She gave a long history of abdominal trouble with seven previous abdominal operations between 1926 and 1951. The original complaint was low right-sided abdominal pain. In 1926 a colopexy was performed and in the next few years two laparotomies were carried out because of continued pain. Then in 1946, an attempt to undo the colopexy resulted in an external ileal fistula which closed spontaneously. Since then she has had another three operations to relieve intestinal obstruction due to adhesions. At the last of these the jejunum and transverse colon were anastomosed but this was performed so high in the small intestine that the patient has since suffered from severe colic and diarrhoea.

The patient had been taking morphine by injection since 1942, and the symptoms produced by the jejuno-colic anastomosis aggravated the addiction. When admitted on 11.4.54, for closure of the anastomosis, the patient's average daily consumption of morphine was 7 grains (450 mg.) in 1-grain (64-mg.) doses. Her maximum had been 12 grains (768 mg.) in 24 hours and she had often taken single doses of 2 grains (128 mg.). Even with this dosage she was able to work in her home and her garden and to drive her car.

On admission, the patient's general condition was fairly good. Mentally, she was very alert, taking a keen interest in affairs. A tall woman, she was thin, but not markedly so, and had lost 42 lb. (19.1 kg.) in the previous three years. The pupils were in a position of mid-dilatation, reacting to light, and she complained of no symptoms due to morphine. The blood pressure was 130/90; haemoglobin 11.3 g. per cent.

For many years, in addition, she had been taking a sedative at night and required a different drug every few months as she rapidly acquired a tolerance towards each in turn. Most of these had been of the barbiturate series.

No record is available of her previous anaesthetics as these were administered in other hospitals.

ANAESTHETIC TECHNIQUE

At 9.30 p.m. on the night before operation, the patient was given sodium barbitone (Medinal) 7½ grains (480 mg.) and promethazine hydrochloride (Phenergan) 50 mg. In addition, morphine 1 grain (64 mg.) was administered at 10 p.m., 1.30 a.m., and 6.30 a.m. The nursing staff reported that she slept at intervals during the night.

At 8.45 a.m., premedication consisting of promethazine hydrochloride (Phenergan) 50 mg., pethidine 100 mg., and scopolamine 1/150 grain (0.44 mg.) was injected intramuscularly. On arrival at theatre one hour later, the patient appeared paler than usual, was alert, and expressed the opinion that her injection had not been different from her usual grain of morphine.

Anaesthesia was induced at 9.50 a.m. with 10 ml. of 5 per cent thiopentone (0.5 g.) intravenously. Consciousness was lost after the injection of 4 ml., and the patient stated later that she had become unconscious more rapidly than at previous operations. A large cuffed endotracheal tube was passed orally after the injection of d-tubocurarine chloride 30 mg. and the spraying of the larynx with 1 per cent amethocaine. Anaesthesia was continued with nitrous oxide and oxygen (2 l./min. of each), with controlled ventilation using the Blease Pulmoflator with carbon dioxide absorption. Intravenous pethidine and further doses of d-tubocurarine and thiopentone were given as required.

The operating time was 1 hour 55 minutes and during this period the condition of the patient gave no cause for anxiety. The pulse rate, which was 60/min. prior to induction, did not vary much and at no time did it exceed 70/min. The systolic blood pressure varied between 140 and 160 mm. Hg during anaesthesia (fig. 1). When the operation commenced, a slow intravenous infusion of 5 per cent dextrose in water was given and 40 minutes later this was followed by 500 ml. of blood transfused slowly.

At the conclusion of the operation, decurarization with atropine sulphate 1/100 grain and neostigmine methyl sulphate (Prostigmin) 2.5 mg. produced a satisfactory respiratory excursion. The total doses of drugs used during anaesthesia were thiopentone 0.75 g., d-tubocurarine chloride 70 mg., and pethidine 125 mg. A few minutes after the endotracheal tube was withdrawn the patient showed signs of regaining consciousness. One hour later she was asking for morphine.

DISCUSSION

The outstanding feature of this patient, from the point of view of the anaesthetist,

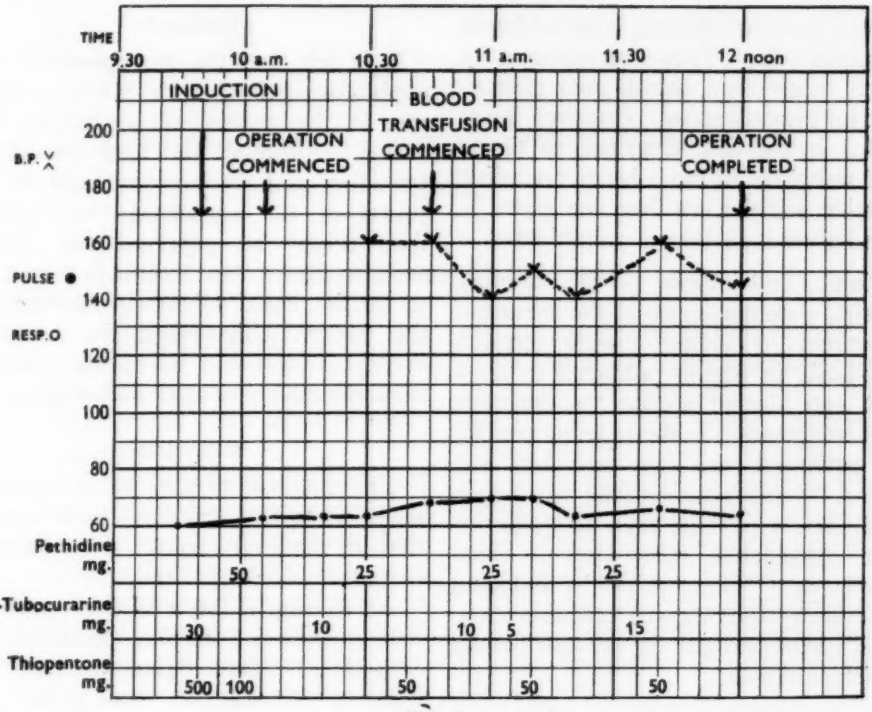

FIG. 1

was the high degree of tolerance shown to morphine and barbiturates. As an illustration of this tolerance, the patient, on the 6th night after operation, had morphine 1 grain (64 mg.) at 9.30 p.m. and at 2.30 a.m.; between times, she was given, first, sodium barbitone (Medinal) $7\frac{1}{2}$ grains (480 mg.) orally and then sodium phenobarbitone (Sodium Gardenal) 3 grains (192 mg.) intramuscularly, yet she spent the night reading the previous day's newspapers and completing her income tax returns.

To counter the drug resistance, premedication similar to that used by Baxter, Bolster and McKechnie (1954) during their investigation of phenothiazine derivatives was employed except that scopolamine was substituted for atropine for the additional hypnotic effect. Promethazine hydrochloride (Phenergan) was used for its potentiating action on the anaesthetic drugs, its hypnotic action, and its effect of reducing metabolism.

The anaesthetic technique was that normally used for major abdominal surgery except that slightly more pethidine and thiopentone were given than for routine cases. The result was satisfactory; the patient's condition during the operation was good and there were no post-operative sequelae associated with the anaesthesia.

Macintosh and Bannister (1952) have stated that morphine addicts are not more difficult to anaesthetize than normal

patients, provided the drug is not withheld pre-operatively. They recommend a slightly larger than normal dose of morphine for premedication. However, it was decided in the case described to try to avoid morphine in the 3 hours preceding operation and the last dose was given $3\frac{1}{2}$ hours before induction of anaesthesia.

Minnitt and Gillies (1948), quoting Johnson, state that many of the abnormal phenomena occurring during general anaesthesia in persons addicted to morphine and alcohol are due to defective tissue oxygenation in the nervous system. The patient in the case described was given a 50 per cent mixture of oxygen which should be more than adequate for tissue oxygenation even after 2 hours anaesthesia. Promethazine, by reducing metabolism and oxygen requirements, would further protect the nervous system from oxygen lack.

An attempt was made by Abreu and Emerson (1939) to support experimentally clinical opinions that morphine addicts were relatively insusceptible to anaesthetics and that alcoholic patients were difficult to induce. They found that mice habituated to alcohol were significantly more resistant to induction with ether than were saline-treated controls. However, with mice treated daily with morphine there was a significant decrease in resistance.

Recently, Dundee, Gray, Mesham and Scott (1953) found a morphine addict resistant to their pethidine, promethazine and chlorpromazine (Largactil) "cocktail". Autonomic blockade was incomplete and there was only a slight fall in rectal temperature.

COMMENT

It is felt that the pre-operative promethazine hydrochloride played a major role in the anaesthetic technique described. Although the total amount of pethidine and thiopentone used in the course of the anaesthesia was greater than might be expected for normal patients, the increase was not in proportion to the increased tolerance exhibited by the patient. She stood the operation extremely well and no unusual reaction occurred during or after anaesthesia.

SUMMARY

The medical history of a morphine addict is briefly described with illustrations of the high degree of tolerance to morphine and barbiturates.

An account of the anaesthetic management is given and the total amount of each drug used is stated. Promethazine hydrochloride (Phenergan) was given pre-operatively.

Literature referring to anaesthesia for morphine addicts is reviewed.

It is believed that promethazine hydrochloride played a major role in the successful management of the anaesthesia.

ACKNOWLEDGMENTS

I wish to thank Mr. W. Ian Gordon, under whose care the patient was admitted, for permission to publish this report and his interest in it. I am also grateful to my senior anaesthetic colleagues for their encouragement and advice.

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NON-REBREATHING TECHNIQUES

Simple Adaptation of Standard British Apparatus

BY

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INTRODUCTION

THERE are two standard types of non-rebreathing circuit for nitrous oxide-oxygen anaesthesia.

The first has been available for many years and is obtained with an intermittent flow type of apparatus (McKesson, Walton), a little positive pressure being used to give a continuous flow of gases. A uni-directional gravity check valve incorporated in the apparatus prevents rebreathing unless a reservoir bag is cut into the circuit. Artificial respiration cannot be used without changing to a semi-closed circuit.

The second is a more recent introduction and consists of a standard semi-closed circuit with the addition of a special non-rebreathing valve which is a uni-directional valve adjacent to or incorporated into the expiratory valve close to the face-piece. Artificial respiration by bag-pressure can be used.

Non-rebreathing also occurs in an Oxford Vaporizer where a uni-directional rubber flap valve lies close to the expiratory valve; and when inspiratory-expiratory valves are used for intermittent positive pressure artificial respiration.

Non-rebreathing technique in paediatric anaesthesia with specially designed non-rebreathing valves has been fully reported in the U.S.A. by Leigh and Kester (1948), Slater and Stephen (1951, 1952), Adriani and Griggs (1953), Griggs, Adriani and Benson (1953), Smith (1953) and others. Its value in paediatric anaesthesia is established in the United States, owing to the small respiratory volumes in infants, and requires no further comment.

There has been no British report.

A simple means of converting standard British apparatus to non-rebreathing technique follows.

ADVANTAGES OF NON-REBREATHING TECHNIQUES

In non-rebreathing technique the patient inhales only fresh gases from the machine; all exhaled gases pass directly out of the expiratory valve. The only rebreathing possible is from deadspace in the apparatus. Most of this is under the face-mask and is unavoidable, unless the gases are administered through an endotracheal tube, when the total deadspace may be less than physiological.

There are therefore these possible advantages over a semi-closed technique:

(1) "Expired" gases are not inhaled.

(2) The inhaled mixture is identical to that being delivered from the flowmeters, there being no nitrogen, water-vapour,

nor carbon dioxide to alter the concentrations.

(3). Respiration and anaesthesia are not complicated by an artificially high concentration of carbon dioxide in the inhaled mixture.

(4) The concentration of nitrous oxide and oxygen in the inhaled mixture can be more rapidly changed; this gives more precise control over oxygenation and depth of anaesthesia.

(5) Saturation with nitrous oxide and elimination of nitrogen are more rapidly achieved.

(6) A continuous record of the patient's minute respiratory volume is obtainable for charting (see below). This is of particular value in teaching and clinical investigation, and serves as a simple check on the adequacy of a patient's breathing before return to the ward, if relaxants or analgesics have been used.

British anaesthetists interested in using non-rebreathing technique with existing standard apparatus may do so as follows.

ADAPTATION OF CIRCLE ABSORBER TO NON-REBREATHING CIRCUIT

To obtain a non-rebreathing circuit using a standard continuous flow apparatus such as Boyle's, with a standard circle absorber (e.g. Coxeter-Mushin, Boyle Mk II), carry out one of the two following manoeuvres, which take only a few moments.

Method I. The simpler and quicker.

Use a circle absorber as a semi-closed circuit, with soda-lime turned off, expiratory valve at face-piece open, and a flow of about 8 l./min fresh gases; now place a clamp, such as a sponge forceps, on the expiratory hosing near the face-piece.*

On the Marrett apparatus simply remove expiratory hosing from port, insert stopper, and reconnect.

Method II. Has the advantage that only a single length of hosing is in use.

(1) Remove both lengths of hosing, with connections, from the absorber and place aside, as they are not used.

(2) Block the expiratory port with one of the following†: a cork, a rubber bung from a standard saline bottle, an endotracheal adaptor with a clamp across the rubber portion.

(3) Take a complete semi-closed (Magill) attachment and plug into inspiratory port.

(4) Turn bag control to "shut".

(5) Open expiratory valve at face-piece.

(6) Run in about 8 l./min fresh gases.

By either method inspiration is entirely from the reservoir bag,‡ which contains only fresh gases; expiration is entirely through the expiratory valve. The unidirectional valve at the inspiratory port acts as a gravity check valve.

There should be sufficient tension on the expiratory valve to allow the reservoir bag to fill, if the outlet is occluded.

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* On the Coxeter-Mushin absorber (and others) it is also advisable to empty the ether chamber, and turn the ether control fuil on. This diverts the fresh gases into the reservoir bag which otherwise pass along the inspiratory hosing during the pause between expiration and inspirat'on. This manoeuvre is essential if an M.R.V. reading is required.

† If this is not done the apparatus behaves as a "draw-over" circuit for ether (or Trilene) placed in the ether chamber, to which N_2O, O_2, CO. may be added. When a high flow of gases is used a non-rebreathing circuit is obtained, but if the patient takes a sharp inspiration air enters the circuit through the open expiratory port.

‡ "Reservoir bag" refers to bag in absorption circuit.

A change to a semi-closed circuit is instantly made by turning the bag control of the Magill attachment to "open" in method II; or, obviously, by removing the clamp in method I.

It is possible to carry out artificial respiration by bag pressure, but a valve which can be completely closed digitally during the inspiratory phase is required, e.g. Salt valve, and method II used.

A supply of fresh gases equal to or exceeding the patient's minute-respiratory-volume must be used.

CONTINUOUS RECORD OF MINUTE-RESPIRATORY-VOLUME (M.R.V.)

If the fresh gas-flow exceeds the patient's M.R.V. the reservoir bag will fill (provided there is some tension on the expiratory valve); if the flow is less than the patient's M.R.V. the bag will empty. If the flow is adjusted so that the bag neither tends to fill nor empty, then the sum of the readings on the flowmeters gives the patient's M.R.V.

This method of observing the M.R.V. is very simple, but no tracing is obtained. There are one or two small practical difficulties.

If the M.R.V. changes suddenly, a little time is taken adjusting the flowmeters and the relative concentrations of N_2O and O_2 are temporarily altered, with a possible change in the plane of anaesthesia; the patient's M.R.V. may sometimes exceed the maximum recordable gas-flow of N_2O and O_2, usually 15 l./min, necessitating the use of the by-passes or a change to semi-closed circuit. For the most part, however, a reading of the M.R.V. can be obtained once every minute for charting.

The use of this circuit at the end of an operation gives a simple check on the adequacy of a patient's breathing when relaxants (tubocurarine, etc.) or analgesics (pethidine, etc.) have been used, and will indicate whether neostigmine or nalorphine should be administered. If an adult's M.R.V. exceeds 7 l. it may be assumed that his breathing is adequate. No CO_2 should be used while the check is being made.

DISCUSSION

The main clinical advantage of a non-rebreathing circuit over a semi-closed circuit lies in the absence of an artificially high concentration of CO_2 in the inhaled mixture, but this is too wide a subject to be discussed here.

During light surgical anaesthesia respiration is less regular than in a semi-closed circuit, as pointed out by Clement (1945) who recommends partial rebreathing. For example, operative stimuli may cause hyperpnoea, which may be followed by apnoea (acapnia). This can be troublesome, and does not occur with a semi-closed circuit.

For a given gas-mixture the plane of anaesthesia tends to be lighter than with a semi-closed circuit, owing to the absence of the slight, but definite, narcotic action of CO_2 normally present in the inhaled mixture in the latter.

The technique is valuable for teaching purposes, or when drugs acting on the respiratory system are being investigated. For example, the following can be clearly demonstrated to students as the visual impression is more obvious, and recordable figures are obtainable; the respiratory depressant action of thiopentone, usually preceded by a short stimulation; the depressant action of pethidine and its

prevention or reversal by nalorphine; the depressant action of quite low (5 per cent to 10 per cent) concentrations of cyclopropane with nitrous oxide and oxygen; the stimulant action of low concentrations of CO_2, and so on.

SUMMARY

The two conventional types of non-rebreathing circuit are described. The possible advantages of non-rebreathing techniques are stated. Two simple ways of converting standard British apparatus to a non-rebreathing circuit are described.

A continuous record of the patient's minute-respiratory-volume is obtainable for charting; it is valuable for teaching purposes, and clinical investigations; it is a simple check on the adequacy of a patient's breathing at the end of an operation.

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CONNECTION SIZES ON BRITISH ANAESTHETIC APPARATUS

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Technician

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THE flexible part of current anaesthetic apparatus is characterized by a multiplicity of joints and connections; twelve mating surfaces are not unusual. Although these connections are normally of four standard sizes, manufacturers vary in their use of these sizes for the different connections; difficulty is commonly experienced in interchanging components supplied by one manufacturer with those of another. The anaesthetist, particularly if engaged in research or development work connected with anaesthetic apparatus, is plagued by the number of different ways these various connection sizes are used.

Tables are given here of connection dimensions for three common anaesthetic arrangements. The data were collected through the courtesy of the various manufacturers, and are published to help those having to fabricate parts of anaesthetic apparatus for research or other purposes.

ACKNOWLEDGMENTS

Ready help in supplying much of the information here was given by the British Oxygen Co. (B.O.C.), Medical and Industrial Equipment Ltd. (M.I.E.), and Airmed Limited (Airmed).

SYMBOLS DENOTING TAPER FITS

		Included angle of taper	Length of taper	Small dia	Large dia
M^m	McKesson male	2° 43′	0.6875″	0.7427″	0.775″
M^f	,, female	,,	,,	,,	,,
C^m	Cagemount male	1° 35′	0.5353″	0.8982″	0.913″
C^f	,, female	,,	,,	,,	,,
F^m	Facepiece male	1° 20′	0.616″	0.9167″	0.913″
F^f	,, female	,,	,,	,,	,,
W^m	Waters male	2°	0.401″	1.178″	1.192″
W^f	,, female	,,	,,	,,	,,

SEMI-OPEN CIRCUIT

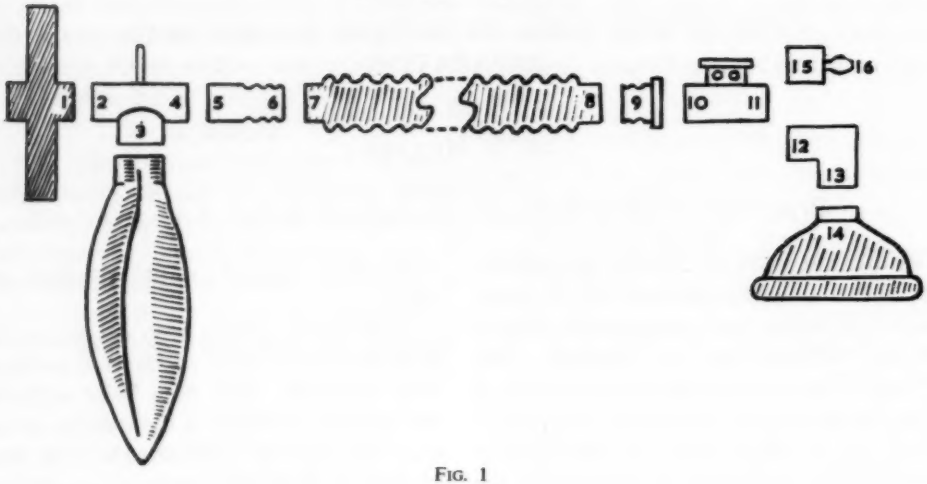


FIG. 1

Diagram number	Fitting	M.I.E.		B.O.C.	
		Internal	External	Internal	External
1	Machine	0.625″	M^m	C^f	—
2	Bag mount	M^f	C^m	0.81″	C^m
3		1″	1.125″	1″	1.125″
4		0.875″	1″	C^f	1″
5	Corrugated tube connector	—	—	0.75″	C^m
6				0.75″	0.937″
7	Corrugated tube				
8					
9	Corrugated tube connector	—	—	C^f	1.03″
10	Expiratory valve	0.875″	0.937″	0.84″	C^m
11		0.687″	M^m	C^f	1″
12	Elbow	M^f	C^m	M^f	C^m
13		0.81″	F^m	M^f	F^m
14	Facepiece	F^f	—	F^f	—
15	Catheter mount	M^f	C^m	0.65″	M^n C^m
16		0.375″	0.605″	0.374″	0.575″

WATERS TO-AND-FRO CIRCUIT

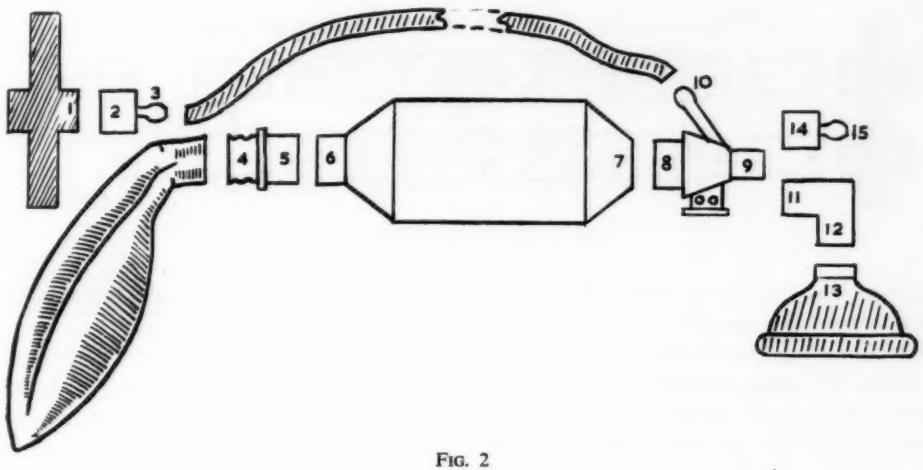


FIG. 2

Diagram number	Fitting	M.I.E.		B.O.C.	
		Internal	External	Internal	External
1	Machine	0.625″	M^m	C^f	—
2 3	Catheter mount	M^f 0.375″	C^m 0.605″	0.65″ 0.375″	M^m C^m 0.575″
4 5	Bag mount	1.187″ W^f	1.25″ 1.25″	— W^f	1.03″ —
6 7	Cannister	— W^f	W^m —	— W^f	W^m —
8 9 10	Expiratory valve and feed mount	1.03″ 0.67″ 0.2″	W^m M^m 0.43″	— C^f 0.2″	W^m — 0.42″
11 12	Elbow	M^f 0.86″	C^m F^m	M^f M^f	C^m F^m
13	Facepiece	F^f	—	F^f	—
14 15	Catheter mount	M^f 0.375″	C^m 0.605″	0.65″ 0.375″	M^m C^m 0.575″

CIRCLE ABSORBER

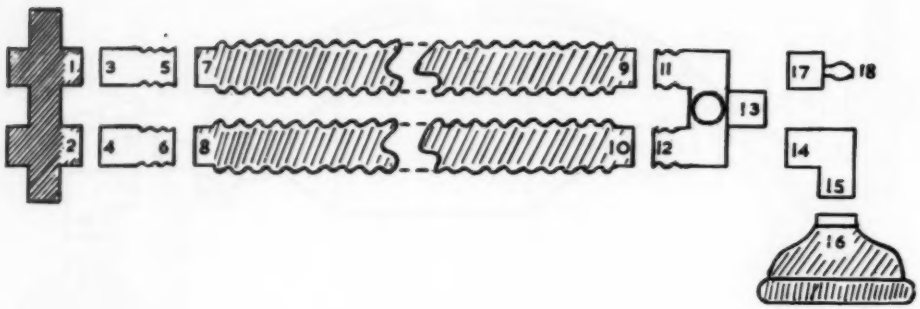


FIG. 3

Diagram number	Fitting	M.I.E.		B.O.C.		Armed	
		Internal	External	Internal	External	Internal	External
1	Machine	—	W^m	C^f	—	C^f	—
2		—	W^m	C^f	—	C^f	—
3	Corrugated tube mounts	W^f	1.25″	0.75″	C^m	0.75″	C^m
4		W^f	1.25″	0.75″	C^m	0.75″	C^m
5		1.15″	1.25″	0.75″	0.937″	0.75″	1″
6		1.15″	1.25″	0.75″	0.937″	0.75″	1″
7	Corrugated tube						
8							
9							
10							
11	"T" piece	0.93″	1″	0.8125″	0.937″	0.872″	1″
12		0.93″	1″	0.8125″	0.937″	—	1″
13		0.59″	M^m F^m	0.75″	F^m	C^f	—
14	Elbow	M^f	0.840″	M^f	C^m	0.781″	C^m
15		0.86″	F^m	M^f	F^m	M^f	F^m
16	Facepiece	F^f	—	F^f	—	F^f	—
17	Catheter mount	M^f	C^m	F^f	—	0.781″	C^m
18		0.375″	0.605″	0.375″	0.575″	0.375″	0.58″

ON CHLOROFORM AND OTHER ANÆSTHETICS

THEIR ACTION AND ADMINISTRATION

BY

JOHN SNOW, M.D.

Licentiate of the Royal College of Physicians

(*Continued from page* 155)

The most convenient position in which the patient can be placed whilst taking chloroform is lying on the back, or side, as he is then duly supported in the state of insensibility, and can be more easily restrained if he struggle whilst becoming insensible. The semi-recumbent posture on a sofa does very well, and there is no objection to the sitting posture, when that is most convenient to the operator. In that case, however, the patient should be placed in a large easy chair with a high back, so that the head as well as the trunk may be supported without any effort, otherwise he would have a tendency to slide or fall when insensible. It has been said that it is unsafe to give chloroform in the sitting posture, on the supposition that it would in some cases so weaken the power of the heart, as to render it unable to send the blood to the brain. Observation has proved, however, that chloroform usually increases the force of the circulation; and although the horizontal position is certainly the best for the patient under an operation in all circumstances, I consider that the sitting posture is by no means a source of danger, when chloroform is given, if the ordinary precaution be used, which would be used without chloroform—that of placing the patient horizontally if symptoms of faintness come on. I have preserved notes of nine hundred and forty-nine cases in which I have given chloroform to patients in the sitting posture, and no ill effects have arisen in any of these cases.

The person who is about to inhale chloroform is occasionally in a state of alarm, either about the agent itself or the operation which calls for its use. It is desirable to allay the patients fears, if possible, before he begins to inhale, as he will then be able to breathe in a more regular and tranquil manner. In a few cases, however, the apprehensions of the patient cannot be removed, and they subside only as he becomes unconscious from the inhalation. It has been said that chloroform ought not to be administered if the patient is very much afraid, on the supposition that fear makes the chloroform dangerous. This is, however, a mistake; the danger, if any, lies in the fear itself. Two cases will be related hereafter in which the patients died suddenly from fear, whilst they were beginning to inhale chloroform, and before they were affected by it; but the probability is that, if they had lived till the chloroform took effect,

they would have been as safe as other patients who inhaled it. If chloroform were denied to the patients who are much afraid, the nervous and feeble, who most require it, would often be deprived of its benefits. Moreover, the patients would, either be prevented altogether from having the advantage of surgery, or they would be subjected to the still greater fear of the pain, as well as the pain itself; for whatever undefined and unreasoning fears a patient may have when the moment comes for inhaling chloroform, he has only chosen to inhale it on account of still greater fear of pain.

Fear and chloroform are each of them capable of causing death, just as infancy and old age both predispose to bronchitis, but it seems impossible that fear should combine with the effects of chloroform to cause danger, when that agent is administered with the usual precautions. Fear is an affection of the mind, and can no longer exist when the patient is unconscious; but the action of that amount of chloroform which is consistent even with disordered consciousness is stimulating, and increases the force and frequency of the pulse, in the same way as alcohol. I believe that no one would assert that a person would die the sooner of fright for having taken a few glasses of wine, or a small amount of distilled spirits, whatever might be the state of his health. When chloroform has been absorbed in sufficient quantity to cause unconsciousness, fear subsides, and with the fear its effects on the circulation. It is a subject of almost daily observation with me that the pulse, which is extremely rapid from some ill defined apprehension, when certain patients begin to inhale chloroform, settles down to its natural frequency after they have become unconscious.

The practice I have always followed has been to try to calm the patient, by the assurance that there was nothing to apprehend from the chloroform, and that it would be sure to prevent all pain; but where it has been impossible to remove the fears of the patient in this way, I have always proceeded to remove them by causing a state of unconsciousness. It would of course be wrong to choose a moment for beginning the inhalation, when fear was producing a very marked depression of the circulation. On feeling the pulse of a gentleman, about twenty-one years of age, in March 1855, who had just seated himself in the chair to take chloroform, previous to having some teeth extracted, by Mr. Thos. A. Rogers, I found it to be small, weak, and intermitting, and it became more feeble as I was feeling it. I told the patient that he would feel no pain, and that he had nothing whatever to apprehend. His pulse immediately improved. He inhaled the chloroform, had his teeth extracted, woke up, and recovered without any feeling of depression. Now if the inhalation had been commenced in this case, without inquiry or explanation, the syncope which seemed approaching would probably have taken place, and it would have had the appearance of being caused by the chloroform, although not so in reality.

MODE OF ADMINISTERING CHLOROFORM

The experiments previously related show that air containing rather less than two grains of chloroform, in one hundred cubic inches, is capable of causing a state of insensibility, sufficiently deep for surgi-

cal operations; but in a creature the size of the human being, an inconvenient length of time would be occupied in causing insensibility with vapour so much diluted. About four cubic inches of vapour, or rather more than five grains of chloroform to each hundred cubic inches of air, is the proportion which I have found most suitable in practice for causing insensibility to surgical operations. In medical and obstetric cases, it should be inhaled in a more diluted form.

Dr. Simpson recommended chloroform to be administered on a handkerchief—the method in which sulphuric ether was administered by Dr. Morton, in the first case in which he exhibited that medicine. The objection to giving chloroform on a handkerchief, especially in surgical operations, where it is necessary to cause insensibility, is that the proportions of vapour and of air which the patient breathes cannot be properly regulated. Indeed, the advocates of this plan proceed on the supposition that there is no occasion to regulate these proportions, and that it is only requisite that the patient should have sufficient air for the purposes of respiration, and sufficient chloroform to induce insensibility, and all will be right.* The truth is, however, that if there be too much vapour of chloroform in the air the patient breathes, it may cause sudden death, even without previous insensibility, and whilst the blood in the lungs is of a florid colour. Chloroform may indeed be inhaled freely from a handkerchief without danger, when it is diluted with one or two parts by measure of spirits of wine, but the chloroform evaporates in largest quantity at first, and less afterwards, until a portion of the spirit is left behind by itself. The process, however, of inhaling chloroform from a handkerchief is always uncertain and irregular, and is apt to confirm the belief in peculiarities of constitution, idiosyncrasies and predispositions, which have no existence in the patient.†

The most exact way in which it is practicable to exhibit chloroform to a patient about to undergo an operation, is to introduce a measured quantity into a bag or balloon of known size, then to fill it up by means of the bellows, and allow the patient to inhale from it; the expired air being prevented from returning into the balloon, by one of the valves of the face-piece to which it is attached. I tried this

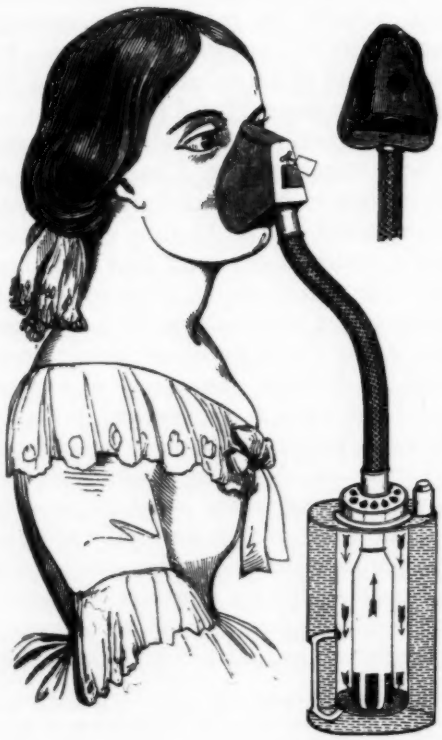
* According to Professor Miller, chloroform was given, at one time, in the Royal Infirmary of Edinburgh, in a somewhat slovenly, and not very cleanly manner; he describes the means of applying it as, "anything that will admit of chloroform in vapour being brought fully in contact with the mouth and nostrils a handkerchief, a towel, a piece of lint, a *worsted glove*, a *nightcap*, a sponge." He says, "In the winter season, the *glove* of a clerk, *dresser*, or onlooker, has been not unfrequently pressed into the service. . . . . The object is to produce insensibility as completely and as *soon* as we can; and there is no saying, *à priori*, whether this is to be accomplished by *fifty* drops or *five hundred*. We begin with generally two or three drachms *spilt* on the handkerchief or lint; and we refresh that, or not, from time to time, as circumstances require." *Surgical Experience of Chloroform*, Edin., 1848, pp. 16 and 17. The italics are not in the original.

† Curious arguments are sometimes used in support of the idea of peculiarities and idiosyncrasies. A medical man informed me, one day, that he had seen a patient inhale an ounce of chloroform without any effect. I expressed my opinion that if she had taken the vapour of one drachm into her lungs within four minutes, or the vapour of a little more than half a drachm within two minutes, she would have been insensible; and that the chloroform had mostly evaporated into the room, without entering the patient's lungs. Then, he said, it would have made all of us insensible. Now to change the air of a moderately sized room of twenty feet square and ten feet high, uniformly with only a grain and a half of chloroform to each hundred cubic inches, so that, if all crevices were closed, a person inside might in course of time, absorb about eighteen minims of the medicine, and be rendered insensible, would require nearly fifteen pounds of chloroform.

plan in a few cases, in 1849, with so much chloroform in the balloon as produced four per cent. of vapour in proportion to the air. The effects were extremely uniform, the patients becoming insensible in three or four minutes, according to the greater or less freedom of respiration; and the vapour was easily breathed, owing to its being so equally mixed with the air. I did not try, however, to introduce this plan into general use, as the balloon would sometimes have been in the way of the surgeon, and filling it with the bellows would have occasioned a little trouble. It seemed necessary to sacrifice a little of absolute perfection to convenience, and I therefore continued the plan which I had already followed.

The great point to be observed in causing insensibility by any narcotic vapour, is to present to the patient such a mixture of vapour and air as will produce its effects gradually, and enable the medical man to stop at the right moment. Insensibility is not caused so much by giving a dose as by performing a process. Nature supplies but one mixture of diluted oxygen, from which each creature draws as much as it requires, and so, in causing narcotism by inhalation, if a proper mixture of air and vapour is supplied, each patient will gradually inhale the requisite quantity of the latter to cause insensibility, according to his size and strength. It is indeed desirable to vary the proportions of vapour and air, but rather according to the purpose one has in view, whether medicinal, obstetric, or surgical, than on account of the age or strength of the patient; for the respiratory process bears such a relation to the latter circumstances, as to cause each person to draw his own proper dose from a similar atmosphere in a suitable time.

The inhaler represented in the adjoining engraving is, with some slight alterations, the same that I have employed since the latter part of 1847. It is made of



metal, and consists of a double cylinder, the outer space of which contains cold water, and the inner serves for the evaporation of the chloroform which the patient is to breathe. Into the inner part of the cylinder there is screwed a frame, having numerous openings for the admission of air, and four stout wires which descend

nearly to the bottom of the space, and are intended to support two coils of stout bibulous paper, which are tied round them, and reach to the bottom of the inhaler. In the lower part of this paper four notches are cut, to allow the air to pass in the direction indicated by the arrows. As the quantity of chloroform which is put in should never fill the apertures or notches, the air which passes through the inhaler meets with no obstruction whatever. There is a glass tube communicating with the interior of the inhaler, and passing to the outside, to enable the operator to see when the chloroform requires to be renewed. The elastic tube which connects the inhaler to the face-piece is three-quarters of an inch in internal diameter, to allow the passage of as much air as the patient can possibly breathe. On the introduction of the practice of inhaling sulphuric ether there was no tubing in this country fit to be breathed through; that in ordinary use was only about one-third, or three-eighths of an inch in diameter,—not more than a quarter of the proper calibre.

The face-piece, to include both the mouth and nostrils, of which that shown in the engraving is one of the modifications, is one of the greatest mechanical aids to the process of inhalation which has been contrived in modern times. Dr Francis Sibson is its inventor. Dr Hawkesley did indeed contrive a very similar one about the same time as Dr. Sibson,—early in 1847,—but he did not make it known. Dr. Ingen Housz made patients inhale oxygen through the nostrils by means of a bottle of India rubber with the bottom cut off; and Mr. Waugh, of Regent Street, had more recently contrived a mouth-piece to be adapted outside the lips, but the usual practice of inhalation previous to 1847, was for the patient to draw in the medicated air by means of a tube placed in the mouth. This led generally to great awkwardness at first, as the patient usually began to puff as if he were smoking a pipe; and it had the further inconvenience, in the administration of ether, that the tube dropped from the mouth, and the patient began to breathe by the nostrils, just as he was getting unconscious. The sides of the face-piece delineated in the engraving are made of thin sheet lead, which is pliable, and enables it to be adapted exactly to the inequalities of the face and the patient can breathe either by the nostrils or mouth, just as his will, or instinct, or other nervous functions, determine.

I have introduced two valves into this face-piece, one which rises on inspiration, to admit the air and vapour from the inhaler, and closes again on expiration, and the other which rises to allow the expired air to escape. I contrived the latter valve to turn more or less to one side, as indicated by the additional line in the engraving, and thus admit more or less of the external air to dilute still further that which passed through the inhaler, and become charged with vapour. By this means the patient can begin by breathing air containing very little vapour, and more and more of the air which has passed over the moistened bibulous paper can be admitted, as the air passages become blunted to the pungency of the vapour.

The object of the water bath is to supply the caloric which is rendered latent, and carried off, as the chloroform is converted into vapour, and thus to render the process of inhalation steady and uniform.

Without the water bath, the evaporation of the chloroform would soon reduce the temperature of the inhaler below the freezing point of water, and limit very much the amount of vapour the patient would inhale; and if the apparatus were warmed by the hand, the temperature would be too high, and the amount of vapour too great. A medical author of great reputation in Paris sent to inquire at what temperature I used the water bath, and being informed, at the ordinary temperature, published his opinion that it had no effect, and might as well be left off. He appeared not to have considered the relations of heat, either to liquids or vapours.

In arranging the bibulous paper in the inhaler, it is my object to contrive that the air passing through, in the ordinary process of inhalation, and at the ordinary temperature of about 60°, shall take up about five per cent. of vapour. This quantity can be diminished, as much as is desired, by turning the expiratory valve on the face-piece a little to one side; and in winter I usually place a short coil of bibulous paper against the outer circumference of the inside of the inhaler, in addition to the central coils which are delineated.

I commonly put two, or two and a half, fluid drachms of chloroform into the inhaler at first. About a drachm of this is absorbed by the filtering paper, and the rest remains at the bottom of the inhaler; and in a protracted operation, when it is seen, by means of the glass tube, that the latter part of the chloroform has disappeared, more is added, by a drachm or so at a time, to prevent the paper ever becoming dry. Mr. Matthews, 8, Portugal Street, Lincoln's Inn Fields, makes the inhaler. There are smaller face-pieces for children. The patient never inhales in so upright a posture as the artist has represented.

There are several other kinds of apparatus in use for the inhalation of chloroform. The most usual consist of Dr. Sibson's face-piece more or less altered, and with a small piece of sponge placed inside. The apparatus which is in most reputation on the continent is that of M. Charrière; it consists of a glass vase with suitable valves, and a fabric for exposing a surface wetted with chloroform to the air which passes through it.

M. Duroy, of Paris, has contrived an ingenious, but very complicated apparatus, which he calls an anæsthesimeter. The object of it is to regulate the amount of chloroform which is inhaled in a given time, and this can be varied from four to sixty drops in the minute; but the experiments which I have related show that the quantity of chloroform employed is not so important as the proportion of it in the inspired air; and although each of these circumstances has considerable influence over the other, in many cases there are conditions in which no regular relation exists between them. For instance, if the inhaler were supplied with sixty drops of chloroform per minute, these sixty drops weigh twenty grains, and produce 15.3 cubic inches of vapour; and if an adult patient were breathing the average quantity of four hundred cubic inches per minute, the air he would breathe would contain nearly four per cent. of vapour, which would answer extremely well; but if the breathing were slow or feeble, or if he should hold his

breath for an interval and commence again, he might breathe air much more highly charged with vapour. Indeed it would depend on the amount of surface moistened with chloroform, the temperature of the air, and other physical conditions, whether or not the air he inhaled might not be charged with chloroform to a dangerous degree; whilst, on the other hand, if the breathing were deep and rapid, as often happens whilst the patient is getting slightly under the influence of the chloroform;—if, for instance, the patient were to breathe at the rate of 1,600 cubic inches, instead of 400, the air he would inhale would contain less than one per cent. of vapour, and he would not become insensible with the utmost supply of the anæsthesimeter, till his breathing should be moderated. M. Duroy also follows the rude and objectionable plan of using a nose clasp, and thus compelling the patient to breathe by the mouth alone.

It is advisable to request the patient to breathe gently and quietly, when he commences to inhale chloroform; in other words, to do nothing but conduct himself as if he were about to fall asleep naturally; for, if he breathes deeply, the vapour feels much more pungent than it otherwise would do, and is apt to excite coughing, or a feeling of suffocation.

In using the inhaler described above, the patient should commence to inhale with the expiratory valve of the face-piece turned to one side, and it should be gradually advanced to the required extent, over the opening it is intended to cover, as the sensibility of the lungs becomes diminished. Not only patients with phthisis or bronchitis, but many sensitive and irritable persons with sound lungs, have a great intolerance of the vapour of chloroform at first, on account of its pungency; and it is necessary to occupy two or three minutes in gradually commencing the inhalation, before the patient makes any appreciable progress towards insensibility. In administering chloroform to children also, it is desirable to begin very gently; by this means, and with a little persuasion, one generally succeeds in getting them to inhale voluntarily; although, occasionally, it is necessary to use a little force to accomplish one's purpose.

(To be continued)

Brit. J. Anaesth. (1955), 27, 196

CARBON DIOXIDE DEPRESSION OF RESPIRATION

BY

E. FALKNER HILL

PROFESSOR PASK (1955) says in his review of cases where postoperative care proved inadequate to meet the circumstances that arose: " It has recently been stressed that a disorder due to the accumulation of toxic amounts of CO_2 in the body, may be associated with the use of drugs which impair the respiratory mechanism—drugs such as depressant anaesthetics or relaxants."

Some twenty-five years ago in a research on the action of procaine injected into the theca (spinal anaesthesia) the accompanying record was made.

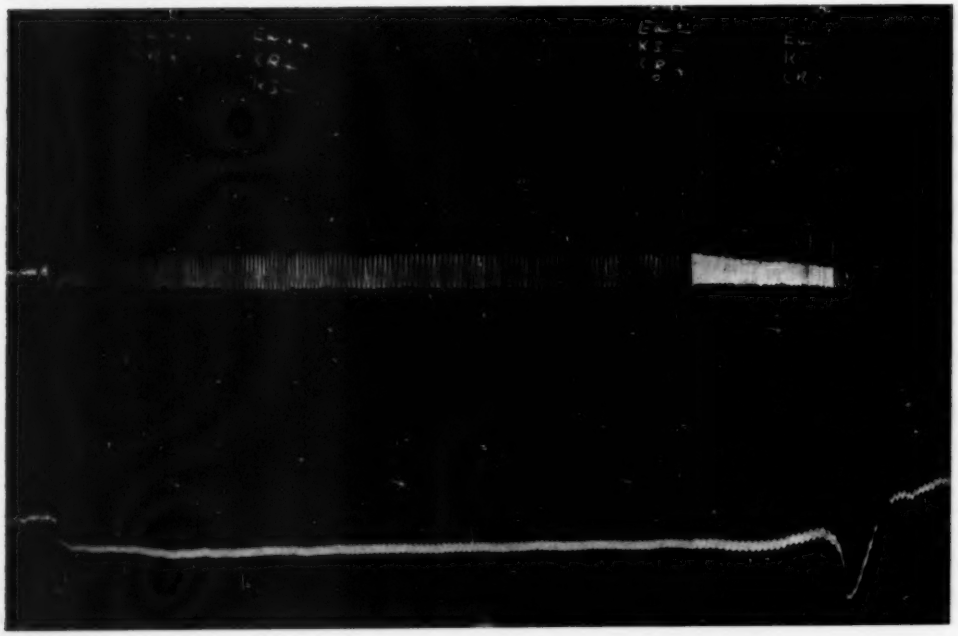
It shows after the injection of 25 mg of procaine in 0.5 ml saline into the theca in the lumbar region of a cat:

(1) Fall of B.P. to 60 mm Hg.

(2) Slowing of the drum twenty minutes later.

(3) Soon after, gradual decline of the amplitude of respiration; at the first arrow distinct slowing of respiration, then normal rate; six minutes later respiration again slowed, second arrow.

(4) Accompanying the decrease in the amplitude of respiration there was a slight fall of B.P., but at the first slow period a



definite but slight rise in B.P. took place.

(5) When respiration slowed again the heart failed, as is evident from the fall of B.P.

(6) Two minutes artificial respiration restored the heart and put the B.P. up to 110 mm Hg.

The explanation of the tracing given at the time was as follows: "A slowing of the respiration produced an asphyxia which, stimulating the vasomotor apparatus, initiated a rise in B.P. which precipitated the cardiac failure. It remains to be seen why the respiratory centre failed. The cat had been given a larger dose of a barbiturate than usual and so the respiratory centre had been rendered less sensitive than it is normally. The increasing CO_2 content of the blood had crept on it unawares, had in fact taken it asleep and poisoned rather than stimulated it. The result of artificial respiration supplies the proof of the accuracy of the foregoing diagnosis. Here again, then, the heart failure is secondary to respiratory failure, but the mechanism of the respiratory failure is somewhat different from those previously seen. It is not due to the procaine but to the depressed state of the respiratory centre due to the overdose of the barbiturate."

It would seem that Professor Pask's opinion as to the cause of some of the deaths of which he writes confirms the accuracy of the explanation of the above tracing given twenty-five years ago, and now appreciated as a cause of death in man.

REFERENCE

Pask, E. A. (1955). *Anaesthesia*, **10**, 1.

BOOK REVIEW

Conduction Anaesthesia (2nd edition). By George P. Pitkin, M.D. Edited by James L. Southworth, M.D., R. A. Hingson, M.D., and Winifred M. Pitkin, M.D. Published by J. B. Lippincott. Price £9.

That posterity should be left with accurate records of the practices of their forefathers is a principle none would deny. Pitkin's mighty volume on "Conduction Anaesthesia" seems to fill this role. The work certainly covers all phases of the subject and is therefore a valuable historical record, and as such will find its way into the cellar shelves of our medical libraries. There is unlikely to be a great demand for such a book because, apart from the high cost and unwieldy size, local anaesthesia has never gained great popularity in this country with either patients or surgeon. The great advance in general anaesthesia in the last few years has given greater comfort and safety to the patient and eased the surgeon's task by giving him better muscular relaxation, diminished bleeding and respiratory movements—a surgical advance which until recently could only be obtained by local or spinal anaesthesia.

From a practical point of view the book might occasionally be consulted by the surgeon who wished for some special reason to perform an operation under local anaesthesia or undertake a therapeutic, or diagnostic, block of the sympathetic ganglia. We do not envy him in his task. The book is verbose, repetitive, inaccurately indexed, although these disadvantages are offset by the excellent illustrations, the number of which contributes largely to the cost and bulk of the volume.

The first five chapters—276 pages—almost a quarter of the book, are devoted to anatomy. In the first chapter the necessity for studying neuro-anatomy is stressed. The authors point out that originally the technique of local anaesthesia simply consisted of infiltration with anaesthetic solution of the tissues within the field of operation. This technique gave way to field blocking in which the solution was injected around the field of operation. Both these methods involved the use of large quantities of anaesthetic solution and limited the surgeon's field of operation to the area infiltrated or isolated by the anaesthetic. These methods, the authors state, have been superseded by nerve blocks and regional blocks. Much less anaesthetic solution is required and the surgeon is free to extend his field of operation if necessary. Nerve and regional blocks require a knowledge of anatomy. The next four chapters are devoted to pure topographical anatomy. The origin, course, and distribution of all the cerebrospinal and autonomic nerves are described concisely and clearly. The text is profusely illustrated by diagrams and dissections. The preliminary chapters on neuro-anatomy are perhaps the best in the book, although it seems to us unnecessary to devote nearly a quarter of the book to pure anatomy, especially as most of it is repeated in the sections on nerve and regional blocks.

Section on nerve and regional blocks. This section is introduced by a useful chapter on the clinical pharmacology of local anaesthetic drugs and general observations on technique. The authors then let themselves go in the succeeding five chapters on technique and application of the various blocks. The section is spoiled by needless repetition. The profuse illustrations seem to provide all the necessary information. The authors quote Confucius as saying, "One picture tells as much as ten thousand words"; they do not, however, follow his advice. The surgeon who wishes to be his own conduction anaesthetist had far better concentrate on the illustrations, which are very clear, consisting largely of a repetition of all the anatomical pictures with the addition of one or more needles. If the surgeon resorts to the text he will probably abandon conduction anaesthesia. The authors solemnly warn the "young block anaesthetist" of the terrible consequences of confusing the motor and sensory nerves to the spleen and blocking the motor nerves instead of the sensory. The consequences of this error, they state, is to produce complete circulatory collapse through dysfunction of the suprarenals. While

still reeling under this astounding statement we read that for a segmental block for surgery of the spleen, paravertebral block of the sixth to the eighth thoracic nerves should be carried out in order to block the afferent and *efferent* nerves. The same bewildering descriptions of straightforward techniques are continued throughout this section. Perhaps the acme of confusing verbosity is reached in their description of blocking the median nerve at the elbow. Apart from advising the injection of 3–4 ml of 2 per cent procaine endoneurally if the operator happens to pierce the nerve, a technique which is rightly condemned earlier in the book, the authors contrive to make a simple technique a most hair-raising procedure, entirely dependent upon hearing clicks as the fascial layers are penetrated, finally injecting their solution between the superficial and deep fascia where they state, erroneously, the nerve lies.

Before leaving this section the reviewer put himself in the place of the "young block anaesthetist" who wanted to block the stellate ganglion. The index refers him to the "scalenus anticus syndrome" on page 910. On turning to this page he would be told to refer to the section on phrenic block on page 870, where he would find Ipparraguire's technique for phrenic block with no reference to stellate ganglion. If not discouraged at this juncture he would search the pages on sympathetic nerve block, eventually finding stellate block on page 897 in the chapter headed "Lumbar, Sacral and Coccygeal Nerves". The authors say that the stellate ganglion can be blocked from an anterior, lateral, or posterior approach, going on to describe, in fact, four approaches, anterior, antero-lateral, lateral, and posterior. Each route is apparently fraught with hazards. The list of quoted complications includes death, cord penetration, haemothorax, and aphonia, and is terrifying. In the reviewer's experience of over 500 stellate blocks, using the posterior route, the only complications encountered have been trivial, consisting of occasional pleuritic pain lasting a few hours and in one case a small pneumothorax which cleared up in a few days.

To sum up this chapter on nerve and regional blocks—the illustrations are good and in most cases self-explanatory. The text is verbose, repetitive, and bewildering.

Local anaesthesia. The next chapter deals with local anaesthesia and on the whole is clearly written and helpful. As in previous chapters, the text is profusely and clearly illustrated.

Topical and refrigeration anaesthesia. The chapter on topical and refrigeration anaesthesia is brief and clearly written. Local refrigeration anaesthesia for poor risk complications has never gained popularity in this country. The principal objection to refrigeration is the necessity of using a tourniquet. There is no doubt tourniquets damage tissue and predispose to oedema of stumps and poor healing.

Spinal anaesthesia. It is quite obvious that the author or authors of this chapter on spinal anaesthesia are persons of experience and know what they are talking about. They are not, however, equally happy in the presentation of their work. They give much useful advice and many techniques they have found satisfactory, but the article is verbose, unbalanced, and at times self-contradictory and, what is perhaps natural, more conversant with American work than British. After a few notes on the history we come to this: "In 1927 at the time that interest in spinal anaesthesia was abating because of fatalities and complications, George P. Pitkin, of New Jersey, a student of Babcock, developed a mixture which he called Spinocain." That is history and correct history and as Evans (1954) points out, "It was Pitkin who revived an interest in spinal anaesthesia both in England and the United States." It is, however, what might be termed external history, it does nothing to touch the true inwardness of the situation. At that time there was great confusion in the minds of the various experts as to exactly how a spinal anaesthetic could cause death, no less than half a dozen different explanations being forthcoming. Thus Koster (1930) says, "Very little is known about the various mechanisms by means of which death is produced." That confusion has been cleared up. The physiological facts upon which spinal anaesthesia should be based have been ascertained. Some reference to these fundamentals should have been made; instead we are treated to a bewildering number of techniques which are good in so far as they are based on these fundamentals and bad when they depart from them. Contradictions appear from time to time. Thus on page 802:

"The onset of surgical anaesthesia after the injection of 0.1 grams of procaine in 2 cc of spinal fluid is about 7 minutes"; yet on page 805 it is said that a suitable dose of amethocaine (a slow-acting anaesthetic), according to the author, will reach the 6th thoracic segment in one minute and in another one or two minutes will reach the 4th. Again, "Nupercaine (1 in 1500) never should be injected in the sitting position." Very sound advice, but on page 833: "The patient remains sitting for a length of time obtained by multiplying the measured length of the back in inches by 2.5 seconds." And this while injecting a solution that rises 20 per cent faster than nupercaine. Still dealing with nupercaine, on page 937: "In large doses nupercaine produces a toxic depression of the heart and cardiac arrest before it produces respiratory arrest." This is a surprising statement and quite contrary to our experience with procaine. As regards the use of spinal anaesthesia in children the authors seem to agree that spinal anaesthesia was frowned on up till 1942 when they quote Vehrs as approving. As far back as 1912 Gray and Parsons in the Arris and Gale lectures have this to say: "Spinal anaesthesia is the most efficient method of inducing a complete afferent block over a large area; and experience has proved that it can be safely successfully and accurately administered to those cases where its administration is called for (particularly toxic children). The younger the child the more urgent is the need for the protection of the synapses likely to be. Finally, should this research incur the accusation of a strong advocacy for the use of spinal anaesthesia in suitable cases, this accusation we will gladly accept." Many of us have been using spinal anaesthesia for children since then and found that the *sine qua non* of success is the establishment of the child's confidence in the anaesthetist.

We are glad to be able to agree with the authors on the following points: (1) It is better to give the spinal anaesthetic before thiopentone or other sedative. (2) It is much better to take blood pressure readings at frequent intervals. (3) "Spinal anaesthesia has become one of the safest and most generally applicable of all forms of anaesthesia if only it is mixed with brains."

Other subjects dealt with are spinal anaesthesia in obstetrics and continuous spinal anaesthesia upon which latter opinions seem to differ. Various sequelae are referred to but there is nothing new to report here.

The final chapters review therapeutic conduction anaesthesia and the complications of conduction anaesthesia in general. In conclusion one cannot help feeling that this mighty atlas of conduction anaesthesia would be of much greater value if the text had been as clear and as good as the illustrations.

Michael Boyd

FACULTY NEWS

THE Seventh Annual General Meeting of the Faculty, which was attended by 85 members, was held on March 23, 1955, and was followed by the Clover Lecture delivered to an audience of 223 by Professor W. W. Mushin on "Measurement in Anaesthesia". The occasion terminated with the Anniversary Dinner held for the first time in the Great Hall in the Royal College of Surgeons; there were 217 members present. The toast of the Faculty was proposed by Sir Charles Dodd and responded to by the Dean, Dr. Bernard Johnson. Sir Russell Brain, President of the Royal College of Physicians, responded to a toast to the Guests, proposed by Dr. Ronald Woolmer, Vice-Dean of the Faculty. Among the many distinguished guests present were Lord Webb Johnson, Sir Henry Dale, Sir Harry Platt, President of the Royal College of Surgeons, Sir Arthur Gemmell, President of the Royal College of Obstetricians and Gynaecologists, and Dr. P. W. Logan, Principal of the University of London.

As a result of the ballot for the three new members for the Board of the Faculty the following were elected to represent the Fellows:

Arthur John Wells Beard
Robert William Cope
Archibald Herbert Galley

There was no nomination for a representative for the Members.

CORRESPONDENCE

NORADRENALINE IN TREATMENT OF ACUTE HYPOTENSION

Sir,—If man is to communicate with his fellows to some useful purpose he must be careful in the choice of his expressions. In Mr. E. F. Shanahan's paper in the January issue of the *British Journal of Anaesthesia* the statement is made that the introduction of l-noradrenaline "is" a marked therapeutic advance, yet nowhere in the paper are to be found comparisons with other drugs and he does not produce a control series.

The study is based on 19 patients in the state of circulatory collapse, 7 of whom died; apparently 2 died almost immediately after the drug was given, and another death occurred during the administration with "massive pulmonary oedema". Mr. Shanahan stressed that the patients were "severely shocked" when treatment was begun. He gives no criteria, however, to support his view that the patients were "desperately ill". His conclusion that "collapse is occasioned by a disproportion between the blood volume and the capacity of the vascular bed" must be based on the findings of others. This he does not make clear and he has not shown that he has measured these things in his patients.

The material in this paper is too wide in scope to allow much in the way of conclusions; it includes collapse alleged to be due to haemorrhage, large bowel obstruction with dehydration, duodenal perforation, and a poor myocardium. He also includes as a cause certain operations, but in these many factors are at work; while the collapse he attributes to a fall in blood pressure makes one wonder whether this is a convenient term like "idiopathic postoperative shock" to cover a lack of knowledge.

More could be learnt from a study which is first limited to one group of patients. The cause of hypotension could then be studied from three points of view, (1) hypotension occurring before operation when the patient is first seen, (2) hypotension occurring for the first time during operation, and (3) hypotension beginning postoperatively. It is only by finding the cause that we can begin to decide on therapy, and Mr. Shanahan's paper has suggested that noradrenaline may be contra-indicated in patients with certain cardiac disorders. If the cause is purely haemorrhage then clearly early transfusion of blood in adequate quantity is indicated. When patients with perforated peptic ulcers are considered, haemorrhage may or may not be an additional factor, as these patients may have had malaena over a long period. It would be difficult to measure the blood volume if the collapse was the type with superficial vasoconstriction, but it might be possible, and any blood in the peritoneal cavity could be aspirated and measured. The time factor is also of importance. So far as I am aware no one has ever measured the "capacity of the vascular bed". Thus the subject bristles with difficulties and much care is needed in planning an investigation of this kind. Unless a careful comparison is made it would be unwise to make a claim for a given method of treatment.

R. P. HARBORD
Leeds

REFERENCE

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Sir,—There are a few points in Dr. Harbord's letter to which I would like to reply.

Firstly, our criteria of "desperately ill" were (1) unrecordable systolic blood pressure, (2) imperceptible pulse in many cases, (3) the presence of a serious intra-abdominal lesion.

Secondly, with regard to the high mortality rate (7 out of 22), only one case died within 1 hour of commencing treatment, not two as he suggests. Of the remaining six, four recovered from their shock but died later from the continuing effect of the original lesion which caused the collapse in the first place. This was clearly pointed out in the paper.

Thirdly, he doubts the efficacy of noradrenaline if the cause of shock is purely haemorrhagic, but three independent groups of investigators, Cappe

and Pallin (1951), Churchill-Davidson (1951), and Livesy and Chapman (1953), apart from ourselves found that resuscitation was more effective in cases of severe haemorrhage with blood and noradrenaline combined than with blood alone. This again was clearly stated in the paper.

Fourthly, he states that haemorrhage may be an additional cause of shock in perforated peptic ulcer. Now perforation and haemorrhage may occur simultaneously but this is very rare indeed. The reason for this is that in the duodenum (and duodenal perforations outnumber gastric perforations in the ratio of 10 : 1) the lesion which causes haemorrhage is the posterior ulcer, whereas the lesion which perforates is the anterior ulcer. In the course of operating on or assisting at over 100 perforations I have never seen more than a trace of blood in the peritoneal cavity. In the vast majority of cases there was none whatever.

Admittedly, we had no control series, nor had we any comparisons with other drugs. Nevertheless, prior to the introduction of noradrenaline we had used such drugs as methedrine and coramine. The effect of these drugs was in no way comparable to the immediate and effective response to noradrenaline as shown in table I of the paper.

E. F. SHANAHAN
Liverpool

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LIVER FUNCTION AFTER SURGERY

Sir,—I have read with interest the paper by Burnett and Cohen (*Brit. J. Anaesth.*, 1955, **27**, No. 2, p. 66) on the cholinesterase activity in the serum of patients following operation. In 1946 with Morand (*Presse méd.*, February 1946, p. 106), Morand and Tanguy (*Acad. de Ch.*, March 1946, p. 261) and with Bathias (*Sem. Hôp. Paris*, February 1949, No. 13, pp. 548–51), we studied the variations of the enzyme activity in the postoperative phase. Our findings were the same as those of Burnett and Cohen, only our conclusions differ slightly because we consider, on the grounds of experimental and clinical data, that the activity of this enzyme is related to the function of the reticulo-endothelial system. It is our opinion that it is this system which controls the plasma level of the enzyme in the liver. The liver, although rich in reticulo-endothelial tissue, is not the only organ which possesses it. Splenectomy results in lowered cholinesterase activity of considerable degree and duration although tests of liver function remain normal. The important role played by the reticulo-endothelial system in the plasma protein is well known. To answer a question put by the authors, we note that the serum cholinesterase activity is considerably reduced by relaxants even if these have not an anti-cholinesterase activity *in vitro*. We consider that they may produce this effect through the mediator of the autonomic system which influences the activity of the reticulo-endothelial system. We have also investigated this possibility (*Acad. de Méd., December* 1947, p. 775).

H. LABORIT
Paris

Brit. J. Anaesth. (1955), **27**, 203

REPORT OF SOCIETY MEETING

THE USES AND ABUSES OF THIOPENTONE*

BY

JOHN W. DUNDEE

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"It is not the fault of the drugs and methods concerned that a syringe and a hollow needle are so susceptible to clinical abuse." (Gillespie, 1950)

THIOPENTONE has been described in such varying terms as a "godsend" and "too dangerous a drug for routine use". Published figures show that in some centres in America it is used in between one-quarter to one-half of all anaesthetics. In Britain thiopentone is more popular and it is safe to say that an intravenous thiobarbiturate is used for the induction of anaesthesia in more than three-quarters of all cases. In view of Gillespie's statement and our ever increasing knowledge of the action of thiopentone, it is timely to consider some of the uses and abuses of this commonly used drug.

USES

Adriani (1946) has admirably outlined the occasions when thiopentone should be used in anaesthesia:

(1) As an anaesthetic for very brief surgical procedures or induction of anaesthesia.

(2) Basal narcotic to supplement nitrous oxide or other more powerful drugs in low concentrations, or in combination with analgesics.

(3) Anti-convulsant.

(4) In hypnotic doses combined with spinal or local anaesthesia.

In as far as the word "anaesthesia", as conceived by Oliver Wendell Holmes, refers to loss of sensation, thiopentone is a satisfactory anaesthetic. Today the more commonly accepted meaning of this word is a state which will permit surgical procedures to be performed without any reaction from, or any dangers to the patient. While thiopentone is one of the best hypnotic drugs known, by modern standards it is an unsatisfactory anaesthetic agent when used alone. It possesses very little analgesic power and the margin between the dose required for relaxation and that which will result in apnoea is very small.

ABUSES

Many of the complications which have followed the administration of thiopentone can be attributed to the terms "short-acting" or "ultra-short-acting" by which it is described in most of the standard books on anaesthesia or pharmacology. A short period of narcosis generally follows the administration of small doses, but large doses may be followed by prolonged sleep. It is slowly broken down in the body, and a cumulative effect is evident if a dose is repeated within thirty hours. Impairment of mental activity often persists for many hours after the administra-

* This is an abridged version of a paper read to the Liverpool Society of Anaesthetists, on October 29, 1954, and the discussion thereon is appended.

tion of thiopentone and the deleterious effects on the cardiovascular system or the liver may last for an even longer period of time. A more descriptive term for thiopentone would be "rapidly-acting diffusible" barbiturate.

Only in very special circumstances should thiopentone be used as the sole anaesthetic, except for very minor procedures. No one would use it alone for major procedures such as gastric resection or the repair of herniae, but it is not uncommon to find it being the sole agent for cystoscopic examinations taking half an hour or more. More anaesthetic is required where the stimulus is strong, as for example during distension of the bladder, or where profound relaxation is required as in reduction and plastering a fracture. Large doses are often followed by delayed recovery. The patient may appear to be lightly anaesthetized when the stimulus is present, but on return to the ward may lapse into deep unconsciousness and be exposed to all the dangers of respiratory obstruction.

Thiopentone is a direct cardiac depressant and, while the intact healthy cardiovascular system can accommodate itself to the effects of small doses, one has no way of telling whether the damaged myocardium can stand up to large amounts. The effects on the cardiovascular system depend on, among other factors, the absolute concentration of the drug in the blood stream. While rapid injection produces a short period of good relaxation, the high concentration of the drug has a profound effect on the blood pressure and cardiovascular system in general. The safety of thiopentone is decreased enormously by a rapid rate of injection.

Insufficient use is made of dilute solutions (2–2.5 per cent). With a dilute solution the rate of injection cannot be so quick, the drug will be more diluted in the blood stream and the effects on the heart will be less. It is interesting to note that no severe damage to a limb has been reported following the intra-arterial injection of a 2.5 per cent solution of thiopentone. This does not mean that the precautions taken to avoid this catastrophe when using 5 per cent solutions should be relaxed with the more dilute solutions. Less severe damage is also likely to follow the extravenous injection of a dilute solution of thiopentone.

Judged by views expressed in the literature there seems to be an improper understanding of the contra-indications to the use of thiopentone and the factors which render patients sensitive to the drug. In table I are listed the contra-indications

TABLE I

Contra-indication to thiopentone reported in 28 publications during the past 10 years

Contra-indication	No. of appearances in literature		
	Absolute	Relative	Total
Myocardial weakness ...	16	4	20
Liver dysfunction ...	14	5	19
Factors interfering with airway	16	2	18
Gross respiratory disease with dyspnoea	15	3	18
Oedema of glottis ...	16	1	17
Shock	9	7	16
Renal disease	9	4	13
Respiratory obstruction	8	2	10
Children under 10 ...	5	4	9
Feeble elderly patients	5	4	9
Severe anaemia	8	1	9
Sepsis	3	3	6
Diabetes	2	4	6
Advanced malignant disease	3	2	5

which appear most frequently in 28 publications on the drug during the past 10 years. In all, 29 conditions were listed in which at least one writer advised against the use of thiopentone. One author lists idiosyncracy to the drug as a contra-indication to its use, but fails to mention how this condition can be detected before the induction of anaesthesia.

It is not a very great exaggeration to say that the absence of suitable veins is the main contra-indication to the use of thiopentone. It is better avoided in patients suffering from porphyria and Addison's disease, or in severe uraemia. Most of the conditions listed in table I result in patients being sensitive to thiopentone, but it can be safely administered in reduced dosage. Another series of conditions stated as " contra-indications " can be grouped under " inadequate airway ", whether this is present before induction of anaesthesia or is liable to occur during the operation. These conditions or procedures are no more a contra-indication to the use of thiopentone than to any other general anaesthetic agent. If a good airway is established before the induction of anaesthesia or steps taken to see that it will remain patent during the operation, then thiopentone can be administered with safety. As an example of this is the report by Williams and Guribruck (1943) of twenty administrations of the drug to patients suffering from Ludwig's angina in which no fatalities attributed to its use occurred. Where the subject was using the accessory respiratory muscles, an endotracheal tube was passed under topical anaesthesia or a tracheotomy was performed before the induction of anaesthesia.

Liver or kidney dysfunction are the next most commonly mentioned contra-indications to the use of thiopentone. Recovery from small doses occurs by diffusion to non-nervous tissues and is independent of the liver, but with large doses the detoxicating mechanism is important in recovery and prolonged narcosis may occur in the presence of severe hepatic dysfunction. Apart from this, it is not advisable to administer large doses of a hepatotoxic agent to a patient with an already damaged liver. The kidney only excretes minute doses of unchanged thiopentone and there is no evidence that its breakdown products, whose excretion may be impaired in kidney disease, have any narcotic properties. However, uraemia which is a common accompaniment of renal disease is one of the most important factors met in clinical practice which prolongs narcosis with thiopentone.

Myocardial weakness is a relative contra-indication to the use of thiopentone. In severe cases it is better to avoid the use of the drug if possible, but where necessary small doses of a dilute solution can be injected very slowly. In this type of case the maximum use should be made of non-toxic adjuvants, especially nitrous oxide-oxygen and relaxants.

The ineffective use of nitrous oxide-oxygen as an adjuvant to thiopentone is only too common. One frequently sees it serving only as a vehicle for carrying a volatile supplementary agent, and playing no major part in the anaesthetic sequence. By using a close-fitting face mask or a cuffed endotracheal tube with high flows of the gas immediately after the induction of anaesthesia a concentration of nitrous oxide in the blood sufficient to ensure

narcosis is soon obtained with a corresponding reduction in thiopentone requirements. Pre-oxygenation before the administration of thiopentone also makes any subsequent administrations of nitrous oxide-oxygen more easily effective. It also permits a longer period of respiratory obstruction should any unforeseen accident occur during the induction period.

There is no justification of the use of thiopentone in outpatients if the patients are allowed to leave hospital unaccompanied. Return of consciousness does not mean that the powers of thought and reasoning are normal; subjects may perform automatic actions, such as walking along a quiet street, but when confronted with a situation which requires a certain amount of quick thinking, such as crossing a busy street, an accident may happen. Further complications may arise from the presence of a period of retrograde amnesia which often follows the use of thiopentone.

The mixing of thiopentone with muscle relaxants, and especially the continued use of dilute solutions of this mixture is a practice which is without any scientific basis. The two types of drugs are broken down by different means and conditions which render patients sensitive to thiopentone often cause resistance to relaxants. In this case an overdose of narcotic can be very easily given. At different periods during an operation either more relaxation or deeper narcosis is needed and, with the use of thiopentone-relaxant mixtures, one cannot be produced without the other.

Mixtures of thiopentone and analeptics are likewise not advised. With Lundy's (1935) mixture of thiopentone and nikethamide one is administering two myocardial depressants to prevent respiratory depression from one of them. Lockett's (1947) suggestion of mixing thiopentone with d-oxyephedrine (Methedrine) also seems unwise, since the latter drug does not effectively counteract the vasodilatation caused by thiopentone, but only "flogs" the heart depressed already by the barbiturate.

The safe administration of thiopentone necessitates a proper appreciation of the pathological conditions which result in sensitivity to it. In anaesthetic practice "shock", anaemia and uraemia are the most important clinical entities in this respect. One has only to recall Halford's (1943) report of its indiscriminate use at Pearl Harbour to appreciate the sensitivity that occurs in injured patients. An "ideal form of euthanasia" was the most complimentary description that he could give to the use of intravenous anaesthesia. Later and more extensive reports from all over the world lead to the conclusion that used judiciously in minimal doses, thiopentone is not in fact necessarily contra-indicated in such patients.

Severe anaemia prolongs thiopentone narcosis (Dundee, 1952), this effect being particularly marked when the haemoglobin level is less than 60 per cent of normal (Haldane standard).

The duration of narcosis is particularly sensitive to alterations in the blood urea level (Dundee and Richards, 1954). With a severe degree of uraemia extremely small amounts of thiopentone will lead to prolonged sleep, and the drug is better avoided.

Other conditions which result in decreased tolerance to thiopentone include

malaria (where anaemia and liver dysfunction may both be present), malnutrition, myasthenia gravis and intestinal obstruction. Where the drug is used to control the convulsions which follow overdose of a local anaesthetic, the dosage should be kept to a minimum to avoid prolonged narcosis. Whether the latter common result is solely due to the thiopentone is open to question.

In conclusion it is interesting to see how thiopentone stands in relation to the requirements of a perfect anaesthetic as outlined by Morrin at the annual meeting of the British Medical Association in 1933, the year before its discovery.

(1) " Administration should be accomplished without discomfort to either the young or old patient and without complicated apparatus." This can be done with thiopentone, but to use the drug without means of inflating the chest or controlling the airway is asking for trouble.

(2) " Induction must be agreeable and the anaesthetic should not have any deleterious effects on the respiration or circulation." Thiopentone is both a respiratory depressant and myocardial poison although these effects can be minimized by proper administration.

(3) " Blood pressure should be maintained at a normal level throughout." One cannot guarantee this, but again the method of administration is of the utmost importance.

(4) " Elimination should be rapid without producing harmful effects on the hepatic, renal or pulmonary tissues." Clinically thiopentone is short-acting after small doses but pharmacologically it is long-acting, irrespective of the dose.

(5) " The anaesthetic effect should be induced gradually and it should be at all times under control." This depends on the method of administration.

(6) " In addition to securing sensory paralysis, complete muscular relaxation should be rapidly and safely obtained." To attempt this with thiopentone is to increase its toxicity out of proportion to the advantages gained.

The writer in 1933 concludes: " The perfect anaesthetic awaits discovery . . . the anaesthetist has welcomed the products of the analytical chemist with faith and hope." We shall never find the ideal anaesthetic; rather we shall aim at the ideal anaesthetic combination. However, we owe a lot to the " analytical chemist " for producing a drug, which, if its limitations are appreciated and if it is handled properly can be one of the safest agents available today.

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DISCUSSION

Dr. G. A. Eason remarked that it was gratifying to find that his clinical impressions of the contra-indications to the use of thiopentone agreed with the scientific evidence presented on the subject. He asked Dr. Dundee if he had any views on hexobarbitone. In his experience it was very useful to " cover " local or spinal analgesia. It is less depressant to respiration, less irritant to the tissues and can be given in a higher concentration.

Dr. H. Garry also voiced agreement with the views expressed on thiopentone. For a long time after its introduction she continued to use hexobarbitone and wondered if it had any place in modern anaesthesia.

Dr. G. J. Rees commented on the remarks concerning the rate of injection of thiopentone. The opener had criticized rapid injection of the drug, but there are equally great dangers associated with too slow an injection. Because of the rapid diffusion of the drug larger doses are required to produce sleep, and the use of large doses is one of the things that Dr. Dundee was criticizing. The term " slow injection " is misleading, and a better description would be a " moderate rate " of injection.

He also doubted whether the absence of reported losses of limbs following intra-arterial injection of 2.5 per cent solution of thiopentone was sufficient evidence to support the view that this concentration could be injected into arteries without danger. One must adopt all the known precautions to avoid intra-arterial injection of thiopentone, irrespective of the concentration of the drug used.

Dr. J. E. Riding expressed the view that thiopentone was the prime factor responsible for regurgitation when it was used in combination with a muscle relaxant in the presence of a full stomach. Morton and Wylie (1951) seemed to blame muscle relaxants for the regurgitation, but O'Mullane (1954) has shown

that the cardiac sphincter is completely competent during full muscular paralysis.

Dr. J. Davidson agreed that it was a dangerous procedure to administer thiopentone to patients who were allowed to go home unaccompanied. He quoted a case in which temporary amnesia occurred following its use.

Dr. S. Lipton suggested that the contra-indications to the use of thiopentone depended on the administrator rather than on the drug itself. In his opinion sepsis below the diaphragm was a contra-indication. Should the patient hiccough the abscess may burst through into the pleural cavity.

The President (Dr. G. R. Hopper), Drs. J. B. Hargreaves, J. M. Marchant, R. J. Minnitt, and B. Solomon, also took part in the discussion.

REFERENCES

Morton, H. J. V., and Wylie, W. D. (1951). *Anaesthesia*, **6**, 190.
O'Mullane, E. J. (1954). *Lancet* **1**, 1209.

ANNUAL MEETING OF THE BRITISH AND CANADIAN MEDICAL ASSOCIATION, TORONTO

The programme for the Section of Anaesthetics at the above meeting, under the Presidency of Sir Robert R. MacIntosh, will be as follows:

Wednesday, June 22.

2.00 p.m. "Physiology and Complications of the Trendelenburg Position." C. Langton Hewer, M.B., M.R.C.P., F.F.A.R.C.S.

2.30 p.m. "Whither Now in Anaesthesia?" H. R. Griffith, *M.M.*, B.A., M.D., C.M., F.R.C.P.(C.), F.F.A.R.C.S., D.A.

3.00 p.m. "Antidotes to Curarizing Drugs." A. R. Hunter, M.D., F.R.F.P.S.G., F.F.A.R.C.S.

3.30 p.m. "A New Technique of Intubation for the Prevention of Contra-lateral Infection in Pulmonary Surgery." Leon Longtin, B.A., M.D., F.R.C.P.(C.).

4.00 p.m. Round Table Discussion of Questions to be submitted from the Meeting.
Chairman: B. C. Leech, *O.B.E.*, M.D., C.M., F.F.A.R.C.S.

Thursday, June 23.

2.00 p.m. "Chlorpromazine." D. A. Buxton Hopkin, M.D., F.F.A.R.C.S.

2.30 p.m. "History of Anaesthesia in Canada." H. J. Shields, B.A., M.B., F.R.C.P.(C.), F.F.A.R.C.S., D.A.

3.00 p.m. "The Anaesthetic and the Anaesthetist." Ronald Jarman, *D.S.C.*, L.R.C.P.(Lond.), M.R.C.S.(Eng.), F.F.A.R.C.S., D.A.

3.30 p.m. "Considerations on Recovery Rooms." Fernando Hudon, M.D., F.R.C.P.(C.), F.F.A.R.C.S.

4.00 p.m. "Anaesthesia for Children and the Newborn." R. W. Cope, L.R.C.P., M.R.C.S., D.A., F.F.A.R.C.S.

4.30 p.m. "Spirometry in the Assessment of Analgesia After Abdominal Surgery." Philip R. Bromage, M.B., B.S., F.F.A.R.C.S., D.A.

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